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QUARTERLY

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EDITORIAL

WHOLE BODY RADIATION IN DIAGNOSTIC RADIOLOGY

Another good contribution has appeared[1] in the immense literature now available on protection in diagnostic radiology. This shows again that in radiography of the chest particularly, the single most important factor in limiting gonadal radiation is collimation of the X-ray beam. This article is commended to the readers of The Journal of the Canadian Association of Radiologists as another reminder about the importance of beam collimation in the protection of patients referred to the radiologist. It is our impression that in spite of many similar studies and reminders, the importance of beam collimation is not taken very seriously by physicians in Canada, and specifically by radiologists. On the contrary, of films referred from many parts of Canada, only a minority show that proper efforts have been made to limit the diameter of the useful beam to the part of the patient's body being studied. This impression gives us particular concern because we see more X-rays of infants and children than of adults and it is in the radiographs of the young patients that care in this matter is so important.

Leaving aside for the moment the still incompletely resolved question of "threshold dose" and "permissible dose", it is surely clear to all physicians having even the remotest interest in radiation hazards that radiation to the young patient should be kept to minimum levels, and that the gonads should be protected whenever possible. If it is indeed clear, it should be a matter of daily concern to the radiologist. If he is not preoccupied in providing proper protection for his patients, what responsible person is? If he is unwilling to take the time and assume the responsibility for this protection, how can he claim special knowledge, how deserve his colleague's respect?

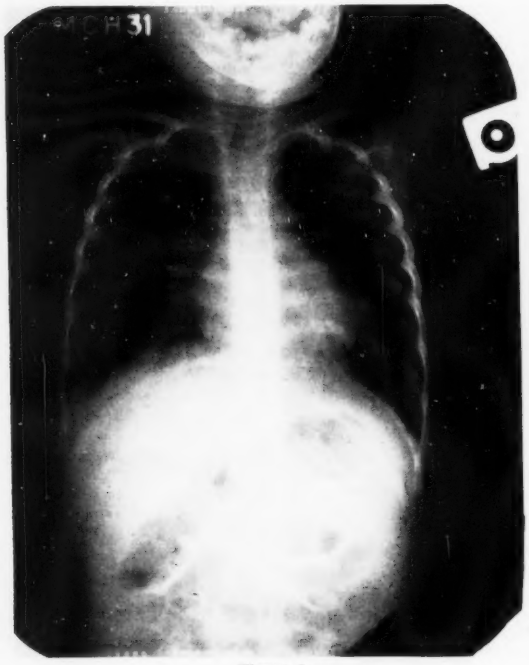

Figure 1

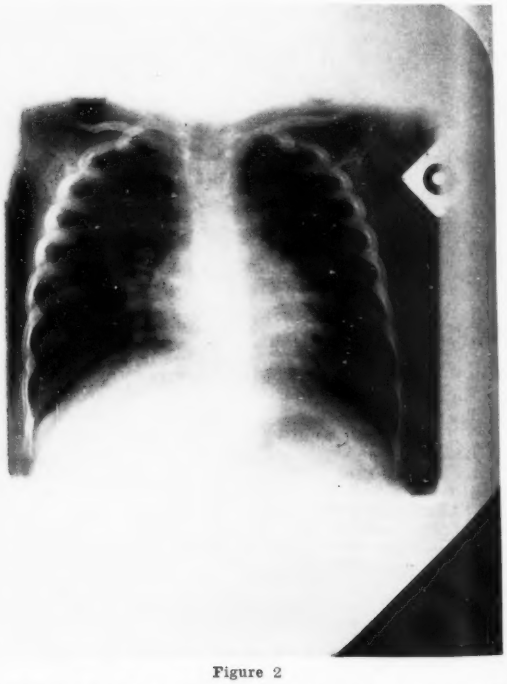

Figure 2

The radiographs were photographed by transmitted and reflected light to show the entire film including its turned corners, with (Fig. 1) and without (Fig. 2) adequate beam collimation.

Figure 1 is the type of chest X-ray of a young child which we see frequently. This figure shows the entire radiograph with its trimmed corners, and the edge of the X-ray beam is nowhere visible. It is our contention that in such a case one is justified in assuming that the young patient has received total body radiation during this exposure. Figure 2 shows a radiograph of the same child's chest, and the edge of the beam is clearly visible, outlining the region essential to the study. We make bold to remind Canadian radiologists of what they know very well already — the radiograph shown in Figure 2 is superior in technical quality because of diminution in the scattered radiation; the patient has been adequately protected during the examination, and the attending personnel have been protected too*. Our experience indicates that this is by no means an unnecessary reminder.

To err is human, but to consistently err in over-exposure of patients, particularly young patients, is perhaps asking too much of even divine forgiveness.

J. S. D.

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1. Epp, E. R.; Weiss, H., and Laughlin, J. S.: Measurement of Bone Marrow and Gonadal Dose from Chest X-ray Examination as a function of field size, field alignment, tube kilovoltage and added filtration. Brit. J. Radiol.; 34; 85-100; 1961.
2. Lewis E. Etter: A New Look at Chest Radiography. Am. Coll. Rad. Bull. Volume 17, No. 7, July 15, 1961, p. 2.

* *The attention of our readers is enthusiastically directed to a recent editorial[2] by Dr. Etter on the economics of beam collimation in adult chest radiography. It comes as welcome news to those of us who have not thought carefully enough about costs, that large amounts of money can be saved by proper attention to reduction in the size of the X-ray beam.*

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PULMONARY HYPERTENSION IN INFANCY AND CHILDHOOD
THE GORDON RICHARDS MEMORIAL LECTURE *

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The Children's Hospital Medical Center
Boston, Massachusetts

Twelve years ago on the 13th of January, Gordon Earl Richards died in the Toronto General Hospital of aplastic anemia. On that day, Canada lost an outstanding citizen and Radiology one of its great pioneers and leaders in the use of roentgen rays and radium in the treatment of cancer. Just a few years before his death, Queens University conferred on Dr. Richards the degree of Honorary Doctor of Laws. The citation reads in part, "A foremost Canadian Scientist in cancer radiotherapy, internationally known as a teacher, researcher, administrator; greatly serving mankind."

This lecture today is given to honor the name and memory of Gordon Richards. As his knowledge was vast and his interests catholic, I am sure that he would have forgiven this break with tradition in that this Memorial Lecture is not on radiotherapeutics.

Pulmonary hypertension has, during the past few years, been extensively studied. In most instances, it is roentgenologically demonstrable and frequently the etiology can be determined, but in this lecture I would like to limit the discussion to those aspects of the problem seen in pediatric practice.

Any pulmonary artery pressure above the upper limit of normal of 30/15 mm. mercury implies pulmonary hypertension. In severe cases it may reach systemic levels. Unfortunately, there is little if any correlation between roentgen findings and the level of pulmonary artery pressure so that by roentgen methods it may be quite possible to state that pulmonary hypertension is present, but it is not always feasible to estimate the severity. By means of a simple formula, pressure equals flow times resistance, one can understand some of the dynamics of the problem.

There are three basic types[1,2]:

1. Passive, due to high pulmonary venous pressure.
2. Hyperkinetic, due to increased pulmonary blood flow.
3. Vaso-occlusive, diminution in the size or number of vascular pathways, and subdivided into:
   (a) Obstructive, due to Agents that reduce the lumen of the vessels.
   (b) Obliterative, reduction of vascular bed from intrinsic disease of the vessels.
   (c) Vaso-constrictive, due to functional contraction of muscular arteries.

Before taking up these in detail, it is necessary to have some small understanding of the nature of the structures with which we are dealing. In the normal muscular artery the form and arrangement of elastic tissue is closely related to intravascular pressure. In a high pressure vessel, fibrils are long, uniform and arranged circumferentially, parallel to each other. In a low pressure vessel, the fibrils are apt to be short and irregular in shape and distribution. In the fetus and the newborn infant, there is high resistance in the pulmonary vascular bed leading to pulmonary hypertension so the blood flows from the pulmonary artery through the patent ductus to the aorta. This is evidently brought about through collapsed lungs as well as through muscular constriction. In the newborn infant with high pulmonary artery pressure, the elastic fibrils are almost aorta-like. As soon as the lungs expand after birth and the ductus is obliterated, there is an abrupt fall in pulmonary artery pressure, and then further gradual diminution until the level approaches approximately 1/5 of the systemic pressure. Once the pressure has fallen there occurs, during the first three to six months of neonatal life, a change in the vascular walls

* *Presented at the Annual Meeting, The Canadian Association of Radiologists, January 24, 1961, Saint John, N.B.*

to resemble that of the adult — a low pressure vessel[1,5]. This sequence of involution may be interrupted or prevented if there is some intrinsic defect in the wall of the vessel itself, as perhaps may exist in so-called primary pulmonary hypertension, or very much more commonly if there is a two-fold or more increase in pulmonary blood flow. One would at first think that the appropriate pulmonary pressure could be maintained by relaxation of the vessels in the presence of an increased flow, but in such an instance there would be a large run off of blood into the lungs leading to a fall in the systemic pressure so that the vessels constrict to maintain a higher than normal resistance. Involution is prevented and the vessels continue to resemble the fetal. It has been called a high-resistance high-reserve vessel. With long continued hypertension, further changes may take place in the vessel wall, eventually leading to the high-resistance low-reserve vascular bed where the large muscular arteries exhibit obliterative intimal changes, and smaller vessels may be dilated and thin beyond obstructive lesions. These changes have been graded one to six[6]. Grades four, five and six corresponding to a high-resistance low-reserve vessel may be quite irreversable even though pulmonary flow is reduced again to normal.

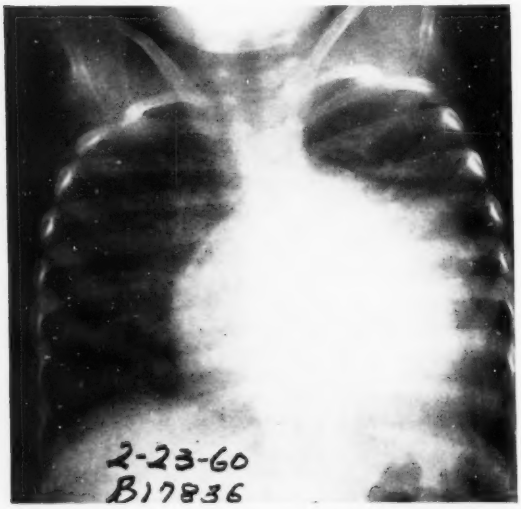

Figure 1 — Mitral atresia with hypertrophy of the right ventricle and atrial parectasis. There is edema of the lungs and hypertension producing enlargement of the main pulmonary artery.

Only very gross changes can be recognized in the pulmonary vessels by roentgenographic means. By careful inspection of the plain films and accurate observation of the behavior and distribution of pulmonary arteries as seen through the fluoroscope or by the various techniques of serial or cineangiography, certain patterns can be detected that will give clues to the nature of the mechanism producing the pulmonary hypertension. Each of the three major types, which will now be considered in more detail, leads to a recognizable pattern that differs from the other. The pattern too may vary somewhat with the age of the patient at the time of onset of the condition.

Common Causes of "Passive" Pulmonary Arterial Hypertension.

1. Anomalous pulmonary venous return:
   (a) Entering thoracic veins but with obstruction.
   (b) Entering below the diaphragm.
2. Cor triatriatum.
3. Tumor of the left atrium.
4. Congenital or acquired mitral valve obstruction.
5. Chronic left ventricular failure:
   (a) Myocarditis.
   (b) Endomyocardial elastosis.
   (c) Underdeveloped left heart syndrome:
      i Coarctation of the aorta.
      ii Aortic stenosis.

In the presence of increased pulmonary venous pressure from any cause there is a compensatory rise in the arterial pressure, and frequently the pressure may continue to rise well beyond the level necessary to overcome the post-capillary resistance. The pulmonary vascular changes associated with hypertension in acquired mitral stenosis have been thoroughly investigated[9,11]. There is segmental constriction of the pulmonary arteries in the lower third of each lung, meandering of many vessels and marked diminution in the rate of blood flow. The pulmonary veins may appear smaller than usual. The vessels to the upper portions of the lungs appear quite normal and exhibit a normal circulation time. Acquired mitral valvular disease is rarely encountered in Pediatric Radiology but there are a number of congenital malformations where the hemodynamics are similar. In congenital mitral stenosis, mitral atresia (Figure 1) or cor triatriatum, there is marked elevation of the left atrial pressure which is promptly reflected in an increase in post-capillary venous pressure. If arterial hypertension is present and significant, the lower third of each lung, if not obscured by congestion and edema, may appear ischemic when compared to the upper two thirds (Figures 2, 3(a)(b)(c)). Septal lines, although frequently seen in older children, can be identified but rarely in infants. Pulmonary hypertension

can rapidly ensue in association with anomalous pulmonary venous return when the pulmonary veins drain into the venous structures below the diaphragm, the obstruction to venous return is usually so great that marked pulmonary edema occurs rapidly before there is any evidence of cardiac enlargement, and the patients will die unless operative correction is instituted promptly. Pulmonary arterial hypertension is always present in a similar situation where the anomalous pulmonary veins are constricted before they enter one of the major systemic veins in the chest.

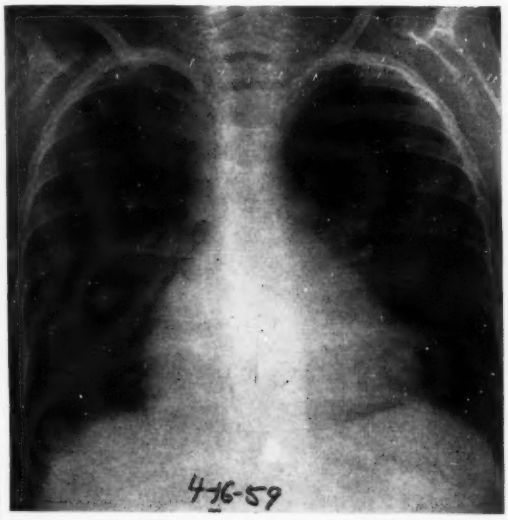

Figure 2 — "Passive" pulmonary hypertension due to chronic myocarditis. The lower third of each lung appears slightly more radiolucent.

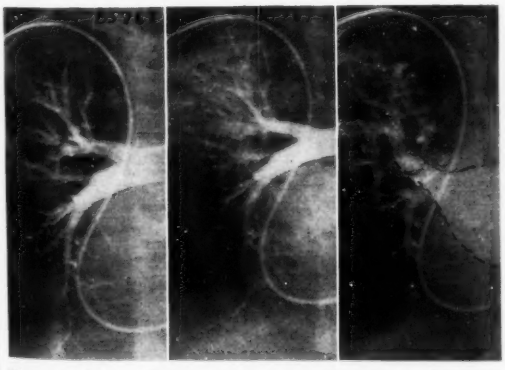

Figure 3(a) — Same patient as Figure 2. Pulmonary arteriogram — vessels in the lower third are constricted.
Figure 3(b) — The finer vessels are tortuous and the lung appears ischemic in the lower third. The opaque medium progresses normally through the upper lung.
Figure 3(c) — There has been little progression of the opaque medium inferiorly. There is a normal capillary and venous pattern in the upper lung.

In these patients, it is usually possible, in addition to the changes already noted, to recognize the gross abnormality of caliber and pattern of these veins. The most striking example that we have seen is one in which there was constriction of the veins before they entered the greatly dilated azygous vein.

There is one other group of lesions that must be considered among those that could well lead to "passive" pulmonary hypertension. They have been called rather loosely "the underdeveloped left heart syndrome." The most important of these are infantile coarctation of the aorta, aortic stenosis and sub-aortic stenosis either with or without endomyocardial elastosis. In these patients, because of an apparent inability of the left ventricle of the newborn infant to cope with the work imposed by overcoming the obstruction to the outflow tract, there is, before hypertrophy of the left ventricle can occur, relative mitral regurgitation, dilatation of the left atrium with increased pressure in the atrium and in the pulmonary veins, most usually with failure and pulmonary edema, but if this does not rapidly lead to the demise of the patient or correction of the defect, then pulmonary hypertension could well follow. Myxoma of the left auricle is very rarely encountered in childhood but it too could produce a similar type of pulmonary hypertension.

Common Causes of "Hyperkinetic" Pulmonary Arterial Hypertension.

1. Congenital Heart Disease with left to right shunt:
   (a) Early — i) Ostium primum interatrial septal defect. ii) Ventricular septal defect. iii) Patent ductus. iv) Aortic pulmonary fenestration.
   (b) Late — Ostium secundum interatrial septal defect.
2. Increased flow through one lung:
   (a) Pneumonectomy.
   (b) Agenesis of pulmonary artery.
   (c) Obliteration of pulmonary artery from extrinsic pressure.

The hyperkinetic form of pulmonary hypertension is considerably more frequent and more important in childhood. In these lesions associated with increased pulmonary blood flow a number of patterns can be encountered. It might be helpful for understanding to examine briefly some experimental work carried out in puppies and in adult dogs[10]. Serial observations were made on the changes in pulmonary artery pressure, radiological and histological features of the pulmonary vessels after pneumonectomy or ligation of a single pulmonary artery. Acute occlusion

of a single pulmonary artery produced only insignificant changes in pulmonary artery pressure, but over the next several days pressure gradually rose and reached in subsequent months higher levels in the puppies than in the adult dogs. Two puppies and one adult dog showed a decrease in pulmonary artery pressure occurring after the initial rise. Pulmonary arteriograms revealed progressive dilatation and tortuosity of the pulmonary artery and major branches with pulsations increased in prominence well out into the periphery of each lung. The very finest

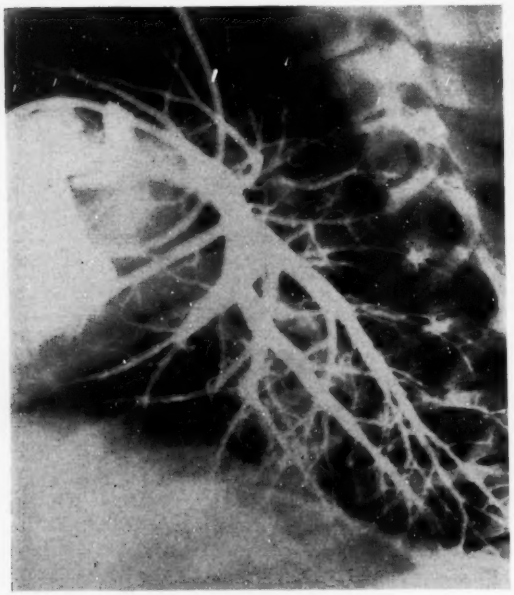

Figure 4 — Shortly after pneumonectomy the vessels in the opposite lung appear normal.

vessels visible on the roentgenographic study appear to be diminished in number and in caliber. While tortuosity of the vessels was a striking feature in the adult dogs (Figures 4 & 5), it appeared very much less prominent in the puppies (Figure 6). It is possible that the response of the pulmonary vasculature is related to the difference in the compliance of the major pulmonary vessels in the young dogs as compared to the adults, even though no significant difference was noted between histological features of the adult and young animals. Medial hypertrophy of the smaller and medium-sized vessels with an increase in the thickness of the wall at the expense of the lumen was present. In these animals, the pulmonary artery flow through the remaining lung would be roughly double at rest. Under conditions of stress or exercise the flow may be four or more times the normal, and it is now well documented that pulmonary hypertension does occur following pneumonectomy. One would suspect that hypertension would develop readily in the occasional very young infant who had had a pneumonectomy or who, at a very young age, suffered obliteration of one main pulmonary artery. It is also possible that the patient born with agenesis of one pulmonary artery will develop hypertensive changes in the other.

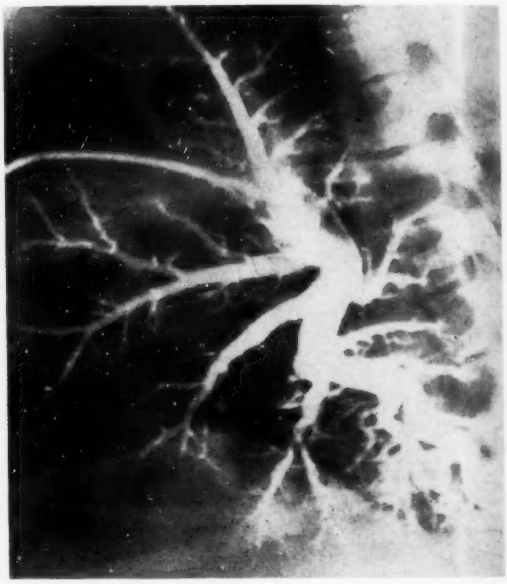

Figure 5 — Same adult dog as Figure 4. Twelve months later there is extreme "Hyperkinetic" hypertension. The vessels are very tortuous.

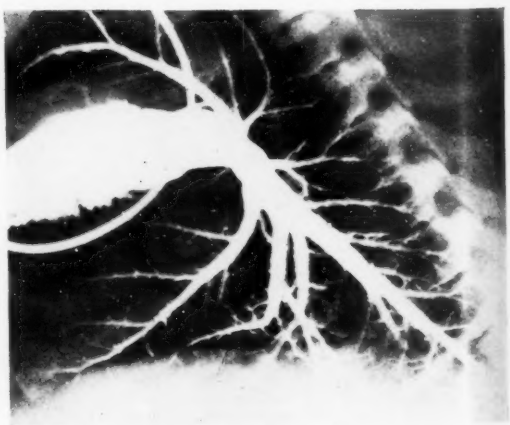

Figure 6 — Nine months following pneumonectomy in a puppy. There is "Hyperkinetic" hypertension but tortuous vessels are not prominent.

As yet we have not had the opportunity to study by cardiac catheterization any of our patients who underwent pneumonectomy before the age of one month or any of the patients with agenesis of one pulmonary artery. It is hoped that this work can be accomplished in the future.

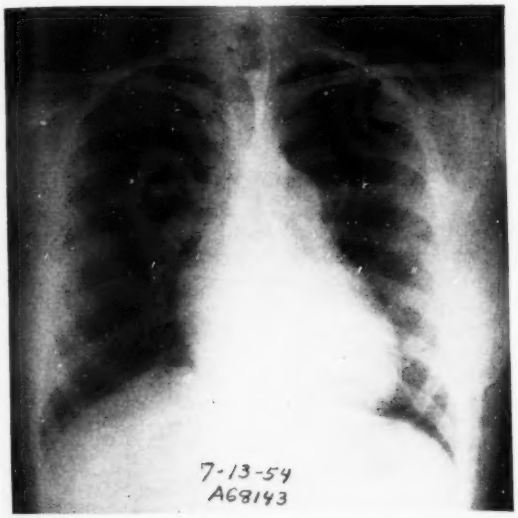

Figure 7 — Ventricular septal defect with pulmonary hypertension. The periphery of each lung is ischemic. The larger vessels are not tortuous.

In a large ventricular septal defect, patent ductus arteriosus, or aortic pulmonary fenestration, the pulmonary flow may be torrential. It has been suggested that this high flow rate prevents the normal involution of the fetal vessels so that increased pulmonary resistance and resultant hypertension may be present from birth. However, in most of these patients, initially, in addition to the usual cardiac changes associated with these abnormalities, one can see that the major trunks of the pulmonary artery may be greatly increased in size with an increase in the expansion of the pulmonary artery and major vessels carried well out into the periphery of the lungs. If severe hypertension follows, the pattern slowly changes with increasing size of the major pulmonary vessels and diminution in the size of the vessels in the mid and peripheral lung fields, giving the so-called pruned or pollarded appearance of the pulmonary arteries. Even in the presence of rather severe hypertension, it is not common to see tortuous vessels (Figure 7). Great tortuosity is, however, a feature of pulmonary hypertension associated with atrial septal defects (Figures 8(a)(b)). In these patients, involution of the pulmonary vessels progresses satisfactorily, presumably because there is no significant increase in pulmonary blood flow during the crucial three or four months of neonatal life. Eventually, there is high pulmonary flow,

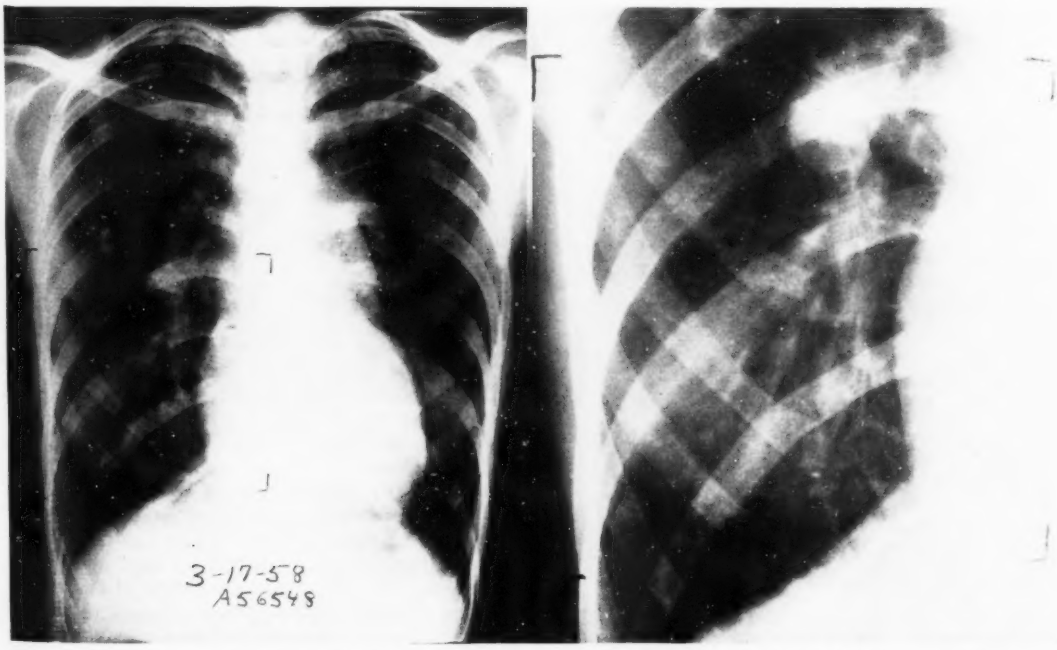

Figure 8(a) and 8(b) — Hypertension due to increased flow from an atrial septal defect. The prominent vessels meander through the lungs.

medial hypertrophy follows and intimal changes occur. The main trunks of the pulmonary artery increase in size and show marked expansile pulsation, and tortuosity of the vessels may be striking. In the later stages, the peripheral vessels, as they do in the ventricular septal defect or patent ductus, diminish in size so that the periphery of each lung appears ischemic. With the development of significant hypertension, the left to right shunt may be reversed and cyanosis develop.

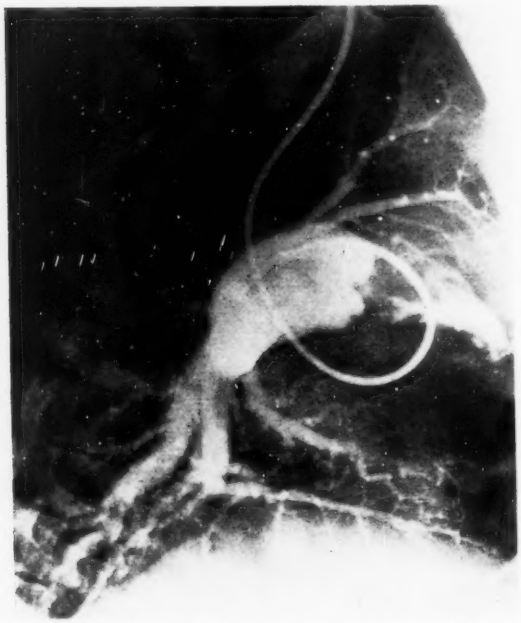

Figure 9 — Thirty minutes after the injection of minute plastic spheres into the pulmonary artery of an adult dog. There is obliteration of the finer vessels. The larger vessels are very tortuous. Tortuosity does not necessarily mean hypertension of longstanding.

Common Causes of "Vaso-occlusive" Pulmonary Hypertension.

1. Multiple congenital stenotic lesions of smaller arteries.
2. Embolization:
   (a) Parasites.
   (b) Indwelling catheters.
   (c) Sickle cell disease.
3. Primary pulmonary hypertension.
4. "Vasculitis" and Collagen diseases.
5. Chronic pulmonary disease with emphysema.
6. Excessive catecholamines?
7. Hypoxia alone? or mixed with any of above.

Pulmonary hypertension due to vaso-occlusive disease is not infrequent in infants and children. Some aspects can be studied experimentally in animals by obstructing the very smallest muscular arteries by introducing minute plastic spheres. Immediately, the major pulmonary vessels increase in caliber, show increase in expansion, and within thirty minutes marked tortuosity of the vessels of adult dogs may be observed. The finer vessels are obliterated and the lung periphery becomes clear (Figure 9).

The vaso-constrictive form may be produced quite readily in dogs by injections of serotonin. When this is done there appears to be constriction of all of the smaller muscular arteries. Only the largest vessels remain unconstricted and these may show increase in pulsation.

The obstructive form is seen in any condition which will produce repeated small embolization of the lungs and is not uncommonly encountered in children with sickle cell disease. The possibility of emoblization with resultant obstructive pulmonary vaso-occlusive disease should be considered in any patient who has had treatment for hydrocephalus that includes a ventriculo-jugular or ventriculo-atrial shunt. Not infrequently thrombi form about the tip of the catheter with resultant embolization. The appearance of the pulmonary vessels varies somewhat with the number of emboli and their size. Minute emboli, lodging in the small peripheral vessels, produce rapidly progressive dilatation of the main trunks of the pulmonary artery with increase in the amplitude of pulsation, tortuous vessels in the mid-lung fields and ischemic peripheral zones. A somewhat similar appearance is produced by a congenital malformation of the vessels with multiple areas of stenosis of the middle-sized and smaller pulmonary arteries.

Obliterative vaso-occlusive disease has been most frequently encountered in the Children's Hospital as a primary pulmonary hypertension which is frequently familial and appears to occur more frequently in girls than in boys (Figure 10). We have had one family with three siblings dying of the disease. On roentgen examination, these patients have shown identical findings. The heart is usually only slightly enlarged with concentric right ventricular hypertrophy. The main trunks of the pulmonary artery are large and pulsate vigorously, but the vessels in the medial two thirds appear normal or even smaller than normal in size with quite clear and ischemic lung peripheries. Early in the onset of the disease, the changes are very difficult to recognize roentgenographically or to distinguish from valvular pulmonic stenosis or from the very rare case of idiopathic pulmonary artery dilatation (Figures 11, 12).

We have had the opportunity to observe a number of patients with pulmonary vasculitis. It is probable that many of these patients in the acute phase of their disease showed areas of transudation or inflammatory reaction in the lung parenchyma, but eventually, as these disappear, one finds a roentgen picture that is identical to that seen in idiopathic pulmonary hypertension (Figure 13).

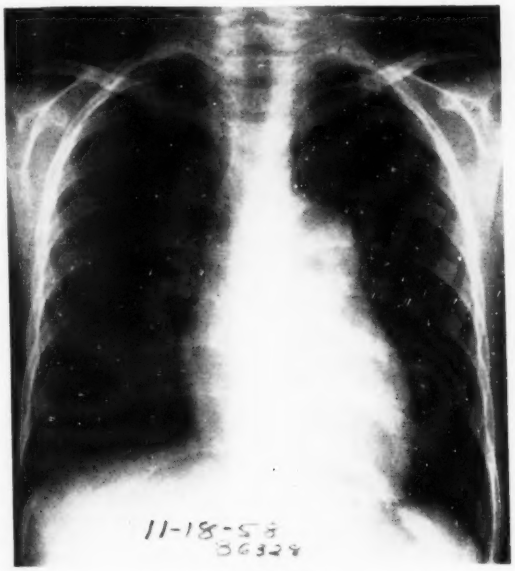

Figure 10 — Primary pulmonary hypertension. The main arteries are very large but the other vessels are greatly diminished in caliber.

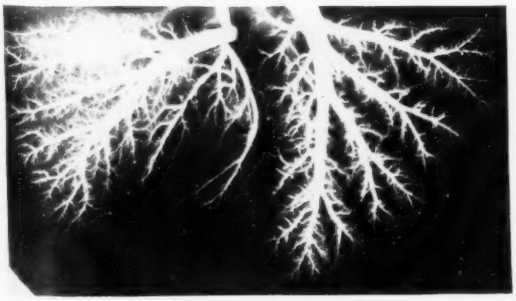

Figure 11 — The normal pattern of the pulmonary arteries in a human.

One would expect the frequent association of pulmonary hypertension with such long-standing pulmonary disease as cystic fibrosis, but such is not the case. Less than 5% of our patients with cystic fibrosis, in spite of extreme emphysema, pulmonary infiltration and bronchiectasis, have shown clinical evidence or roentgenographic signs of pulmonary hypertension. It is likewise rare in the child with even very longstanding asthma or in such deforming conditions as kyphoscoliosis. In the few children studied with severe chronic pulmonary disease in whom hypertension has become manifest, the mechanism appears complex. Hypoxia with resultant increased flow and vaso-constriction certainly plays a part as do fibrosis, atelectasis and actual obstruction of vessels from disease.

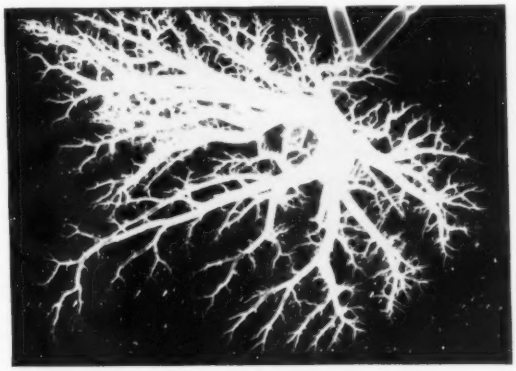

Figure 12 — Primary pulmonary hypertension. The injected specimen discloses little meandering, many of the smaller arteries are obliterated.

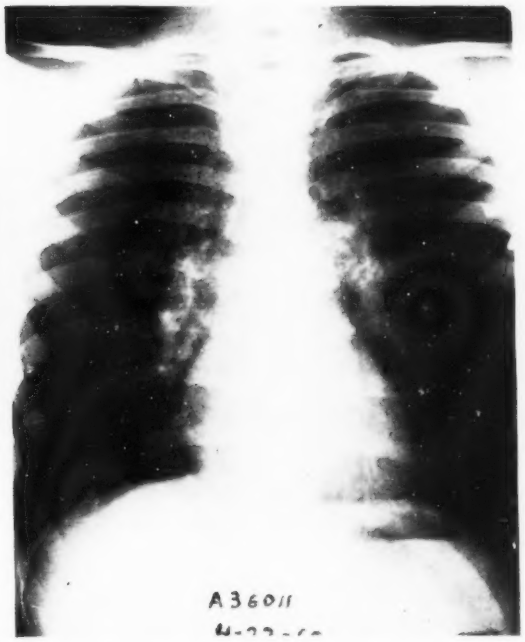

Figure 13 — Ten year-old boy who had severe "vasculitis" six years before. The lungs are ischemic, the main pulmonary arteries prominent, and there is marked hypertrophy of the right ventricle.

One of the classic methods of producing pulmonary hypertension in dogs is by the injection of serotonin or 5-hydroxytryptamine, the chemical that is supposedly liberated in the secretory tumors such as carcinoid or neuroblastoma and sympathicoblastoma. However, in the human being there has not as yet been a truly authentic case reported where pulmonary hypertension has been due to this agent, although pathological changes within the heart have been noted. However, every patient with neuroblastoma should be carefully evaluated for evidence of 5-hydroxytryptamine activity.

I have not observed any patient who because of hypoxia alone developed pulmonary hypertension, although this situation could well occur under certain circumstances. It is well known in animals which, shortly after birth, are removed from lowland pasture to considerable heights for grazing purposes. It is thought that brisket disease occurring in Colorado is the result of diminished oxygen tension with failure of involution of the vessels and persistent constriction of the vessels leading to hypertension and right heart failure, edema of the brisket being the outstanding clinical feature. A newborn infant taken rapidly from sea level to an area of lowered oxygen tension could well develop a similar condition. It is now clear that severe hypoxia produces a stress situation with increased pulmonary blood flow and constriction of pulmonary arteries.

Summary

In each of the three major forms of pulmonary hypertension, passive or post-capillary, or in the hyperkinetic or vaso-occlusive forms, pre-capillary in origin, one can expect to see a differing pattern of pulmonary vessels. The age of onset of the pulmonary hypertension may likewise influence the pattern.

By careful observation of the behavior, caliber and distribution of the vessels and by analysis of associated abnormalities in the heart or lungs, one may detect the presence and suggest the etiology of pulmonary hypertension.

Résumé

On peut s'attendre à trouver un aspect particulier des vaisseaux pulmonaires dans chacune des trois principales formes d'hypertension pulmonaire suivantes: la forme passive ou post-capillaire, la forme hyperkinétique et la forme obstructive, d'origine précapillaire. Le moment où s'installe l'hypertension pulmonaire peut également modifier cet aspect.

Par une étude minutieuse du comportement, du calibre et de la distribution des vaisseaux, ainsi que par l'analyse des anomalies coexistantes dans le coeur ou les poumons, il est possible de déceler la présence et de suggérer l'étiologie d'une hypertension pulmonaire.

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EARLY RADIOLOGIC RECOGNITION OF PUS IN THE JOINTS OF CHILDREN

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Pyogenic arthritis in infancy and early childhood is dangerous to life and dangerous to the future growth and function of the joint. It occurs in the large joints of the extremities, the onset of the disease is often insidious and the clinical signs may be ill-defined and variable.

It has been our impression that radiologic recognition of signs suggestive of pus in the joints is of great value and considerable accuracy. Non-purulent joint fluid collections are uncommon in the first year of life, and the radiologist and surgeon should assume that unexplained joint capsule distention in infancy is purulent until proven otherwise. In order to evaluate this major premise, we have reviewed the radiographs of the first X-ray examination of each of 35 infants and children with suppurative arthritis. The results of this review, together with illustrative case histories and roentgenograms, are the subject of this presentation. A total of 46 joints were affected in these 35 patients.

We have divided the cases into two groups: those under one year of age, who are referred to as "infants", and those over one year of age, classed as "children". This does not correspond either to accepted legal or medical age classification, but has been particularly useful in emphasizing early diagnosis in very young patients.

Clinical Experience

All of the patients at the Montreal Children's Hospital who were diagnosed as having septic arthritis from 1951 to 1960 inclusive have been reviewed. The clinical experience is presented in tabular form in Tables I & II.

The diagnosis was established with certainty in 21 patients, as bacteria were grown from pus aspirated from the joints. In seven others the diagnosis is virtually certain as pus was aspirated and although organisms were not grown on culture, bacteria were found in blood cultures made before or at the time of aspiration. Seven cases without this bacteriological proof have very strong clinical, radiologic and indirect bacteriological evidence of the joint infection, and are included in this series with confidence.

Males outnumber females to a considerable degree (28 males, 7 females), and in the first year of life there were almost as many patients as in the next 15 years (15 infants, 20 children). The hip and shoulder were

TABLE I

35 Patients

Infants *	15	Male	28
Children	20	Female	7

* Patients under 1 year of age.

TABLE II

SEPTIC ARTHRITIS AT THE MONTREAL CHILDREN'S HOSPITAL 1951-1960

	Shoulder	*Elbow*	*Wrist*	*Hip*	*Knee*	*Ankle*
A. *Joints Affected* (Total 46)						
— infants	5	2		14	3	1
— children		3	1	6	10	1
B. *Damaged Joints*						
— infants		2		6		
— children		0		0		
C. *Radiological Observations*						
— soft tissue swelling	5	5	1	20	13	2
— osteomyelitis	3			10	3	1
— capsule distention						
a) fat mass displacement		3		7	12	
b) bone separation	4			14	3	
— osteoporosis				2		

important sites in the infant, while the knee and to a lesser degree the hip were more common sites in the older individual (Table II).

The relative frequency in infants is particularly important radiologically because it may be difficult to make an early clinical diagnosis in this age group. The older child responds to septicemia and joint infection in a more expected manner and the clinical diagnosis in our series was suspected before the initial radiograph. In the infant, septicemia and joint infection may become manifest by irritability, apprehension, failure to feed well, failure to gain weight, vomiting, toxicity, cyanosis, palor, local swelling, muscle spasm, pain in handling, fever, elevation of pulse, or anemia. Sick infants are particularly difficult to handle, and this adds to the problem of clinical diagnosis. Sometimes radiologic examination may have to be done to explore the possibility of suppurative arthritis, and on other occasions it may be a surprise-finding when films are made for other purposes. Approximately half of the infants in this group were first diagnosed from the radiological examination.

None of the children suffered significant joint damage. The infants fared much worse, with two elbows and six hips being permanently damaged (Table II Section B and Case 5). These facts again stress the need for early diagnosis, as delay would likely have increased the number of poor results.

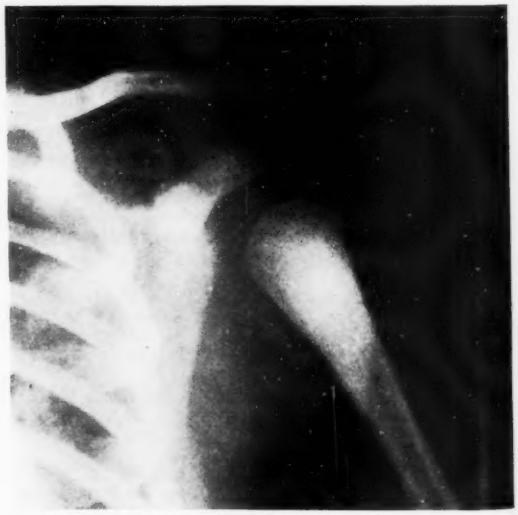
Because of the relatively high incidence and the difficulties in diagnosis of suppurative arthritis in early life, the following case reports concern only infants. All of them were considered normal at birth and were discharged home from maternity hospital well.

Case I: Male, H. R.

This baby boy developed swelling of the right shoulder on the eleventh day of age. He was admitted to hospital four days later because of swelling and induration of the right arm starting at the shoulder and reaching to the elbow. The personal and family history were otherwise noncontributory, except that one member of the family had a sore throat. The hemoglobin was 13.6 grams and the white blood cell count 28,000 with 18,000 neutrophils.

X-ray examination (Figure 1) showed considerable widening of the right shoulder joint, and soft tissue swelling. Suppurative arthritis was diagnosed, and on aspiration 2 ccs. of sero-sanguinous fluid were removed. Culture of the blood grew beta hemolytic streptococci, while pus from the shoulder joint grew no bacteria. The child did well on antibiotic therapy and subsequent X-ray examination showed periosteal new bone formation about the upper end of the right humerus which suggested that osteomyelitis had been present and had initiated the septic arthritis. Likely the ultimate function of the shoulder will be normal, as one month after discharge from hospital there was no abnormality on physical examination. This case cannot be regarded as proven beyond doubt, in view of the failure to recover organisms from the joint fluid.

Figure 1: Case I(a) — Right humeral head widely separated from glenoid fossa and marked soft tissue swelling in the region.



(b) Normal left shoulder.

Case II: Male, B. F.

At age 2 months the baby developed an upper respiratory infection and became irritable. He was seen by a physician and no antibiotics were prescribed, since he did not appear to be ill. Within a week he had become very irritable and had a swinging fever as high as 105°. The left elbow was noted to be swollen and indurated and there was pain in the right hip area when the right thigh was manipulated. The white blood cell count was elevated to 18,000 with 13,000 neutrophils: the hemoglobin was 11.5 grams.

The first X-ray films (Figure 2) showed soft tissue swelling about the left elbow, and posterior displacement of the olecranon fat pad. The right hip joint (not illustrated) was partially dislocated and there was soft tissue swelling in the region.

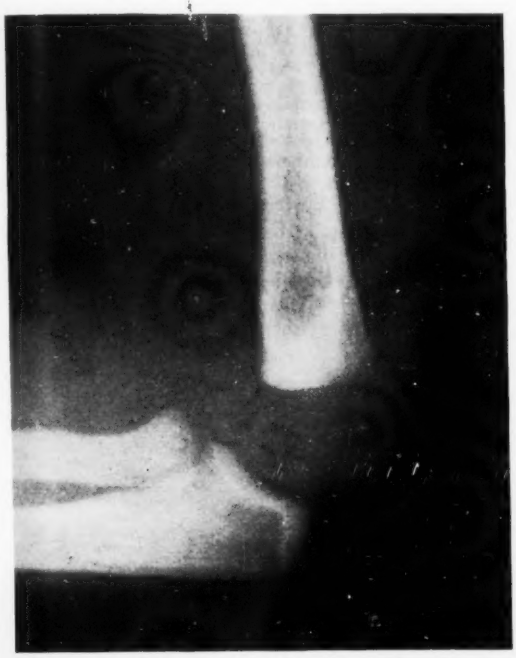
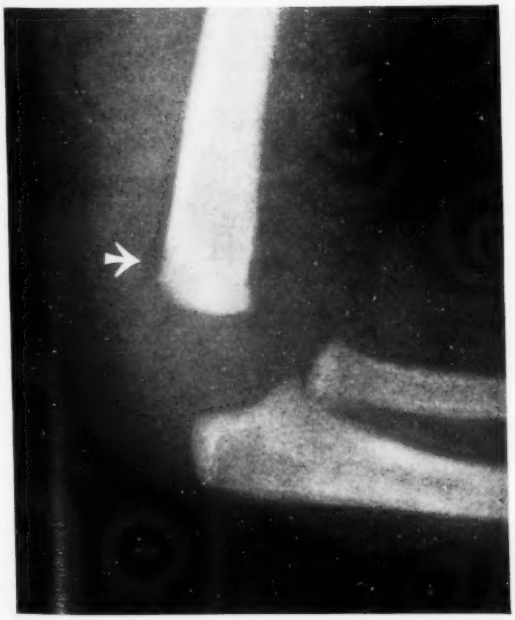


Figure 2: Case II(a) — Normal right elbow.



(b) — Soft tissue swelling surrounding the left elbow and posterior displacement of the olecranon fat pad are marked by arrow.

No bone lesions were demonstrated in the elbow or hip. The diagnosis was septic arthritis in the two joints. Greenish yellow thick pus was aspirated from the right hip and the left elbow joints. Both aspirates and the blood grew staphylococcus pyogenes on culture.

The infant was started on antibiotic therapy on admission. The hemoglobin fell as low as 8.1 grams and responded to transfusion. Cortisone was also used for a short period of time and subsequently the child has done well. The hip joint is normal four months later and the elbow appears normal except for a slight lag in bone maturation and a minor defect in the lower portion of the humerus. It is possible that there may be some permanent damage of the elbow, although the joint now has a full range of movement, and the extremity is used normally.

Comment: In these first two cases the clinical features were suggestive of septicemia and septic arthritis. In each child the diagnosis was made before the radiographs, and the films were of great help in identifying the nature of the disease and indicating that aspiration of the affected joints would likely yield pus.

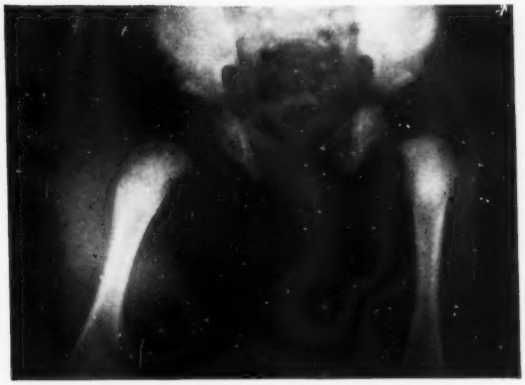


Figure 3: Case III — Four signs are present on the right side; soft tissue swelling of the thigh, dislocation of the hip, subperiosteal new bone formation about the upper end of the femur, and the distended joint capsule displaces nearby fat planes.

Case III: Male, M. M.

At age 27 days the onset of swelling of and failure to move the right thigh was noted. He had a fever and had become irritable. Two days later on admission to the hospital he had considerable swelling of the right thigh and he kept his hip slightly flexed. The skin in the region was red. The hemoglobin was 12.3 grams, and the white blood cell count 15,000 with 9,000 neutrophils.

The radiographs (Figure 3) showed marked soft tissue swelling of the thigh and separation of the bones of the right hip joint. Three ccs. of pus aspirated from the joint grew no bacteria on culture, but material aspirated from the soft tissue swelling nearby grew beta hemolytic streptococci. The child did well on antibiotic therapy, and the follow-up examination six months later revealed a normal joint by physical and X-ray examination.

Case IV: Male, S. B.

This infant failed to gain weight and feed properly. At the age of 11 days he refused all feedings and was irritable. When seen at this hospital at age 13 days he weighed 200 grams less than his birth weight and had a lump overlying the chest wall on the left side which the parents said had been present for two days. He had excessive nasal secretions and his temperature was 102°. There was suspicion that the thigh on the left was larger and firmer than normal. The white blood cell count was 27,000 with 20,000 neutrophils. The hemoglobin was 12.1 grams. No diagnosis could be made from the clinical findings, though septicemia was suspected. It was not until the X-ray film made a day later (Figure 4) that septic arthritis was firmly diagnosed. Then a needle was inserted into the left hip joint and a small amount of pus was aspirated to confirm the diagnosis. The hip joint was incised and 20 ccs. of thick yellow purulent material were sucked out of the region. The chest wall swelling also yielded 4 ccs. of pus on aspiration. No bacteria were cultured from blood or aspirates. The ultimate result will likely be a normal hip, as function is normal and serial radiographs show the joint returning to normal 16 months later.

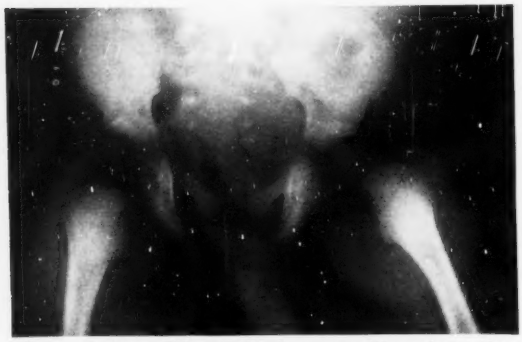


Figure 4: Case IV — Soft tissue swelling about the left thigh and dislocation of the left hip. The fat planes that would indicate capsule distention have been obliterated by edema, and this sign is not useful.

Case V: Female, J. L.

The baby had a "cold" a few days after going home from the maternity hospital. At age 36 days the lower limbs were not used normally and the baby screamed every time they were moved. At age 47 days the baby was seen at this hospital and was admitted. She had not gained weight since birth even though the feeding history was said to be good. All four limbs were partially flexed. A grating noise was heard on movement of the upper limbs and the knees were seen to be swollen and could not be flexed or extended to any extent. The white blood cell count was 15,000 with 10,000 neutrophils. The hemoglobin was 9.6 grams.

The X-ray findings of this child were striking (Figure 5) with destructive lesions of the upper ends of both humerii, the upper ends of both femora, about both knees, and involving the talus of the left ankle. There was soft tissue swelling at all of these sites and the bones of the hip joints and of the left shoulder were separated. As well it was felt that there was fluid in both knee joints and the left ankle joint. Septic arthritis was diagnosed at each of these sites.

Aspiration of the right hip was performed and 2 ccs. of yellow thick fluid were removed. No positive cultures were obtained on this child from the hip or blood. One year later the child has done fairly well except for a partially destroyed left hip joint which remained dislocated and has required orthopedic management and corrective surgery.

Comment: In the last three infants the diagnosis appears obvious in retrospect, from summaries of the pertinent findings on clinical examination, by X-ray examination, and in the laboratory. In the management, however, some of these findings had to be searched for diligently and the diagnosis was not obvious on first examination by a physician. In each infant when the appropriate X-ray examination was performed the nature of the disease became clear. A high degree of suspicion is essential on the part of the referring physician and also the radiologist who interprets the films or who is asked to examine the patient with a puzzling clinical problem.

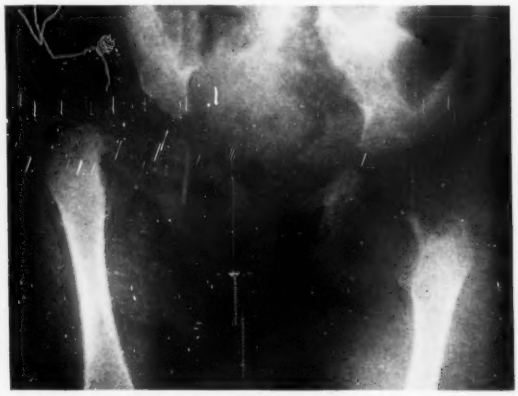


Figure 5: Case V (a) — Both hip joints show osteoporosis, soft tissue swelling, dislocation and destructive lesions of the upper ends of the femora.

Radiologic Abnormalities

The signs that were found in the initial films are listed in Table II under radiological observations Section C.

Soft tissue swelling (Figures 1 - 5) is a very general sign but it does call attention to the abnormal joint. It was found in all of the 46 affected joints and ranged from a subtle minor change to massive swelling of the whole part. Fortunately, in our experience, there was always another radiological sign, except in the one child with osteomyelitis of the wrist.

Osteomyelitis (Figure 5) in the nearby metaphyseal region of the bone is seen in those patients in whom the septic arthritis is secondary to rupture of the inflammatory lesion into the joint space. If present, destruction of a small part of the bone is usually seen in the early film although sclerosis and subperiosteal new bone formation were occasionally present. Eight of the infants examined had osteomyelitis in the first radiograph.

Capsular distention of the joint by pus is very important and it manifests in two manners. There may be *bone separation* (Figures 1, 3, 4, and 5) due to the pressure generated by the inflammatory process. In the shoulder, the humeral head is pushed out of the glenoid fossa; in the hip the femoral head is pushed outwards and then pulled upwards from the acetabular fossa; in the knee, the patella is displaced from the femur. No separation of bone occurs in the elbow, wrist, or ankle due to the strong ligaments of these joints. The patellar displacement is best appreciated when the patella is ossified, although it may be detected before this if there is only moderate surrounding soft tissue swelling. Fortunately *fat mass* displacement occurs in the elbow (Figure 2) and ankle joints and allows detection of capsular distention. The wrist joint is small and tightly confined by stout ligaments and no significant distention of it can be detected radiologically. In the hip joint, capsular distention may be detected by fat plane displacement and or the separation of the bones described above.

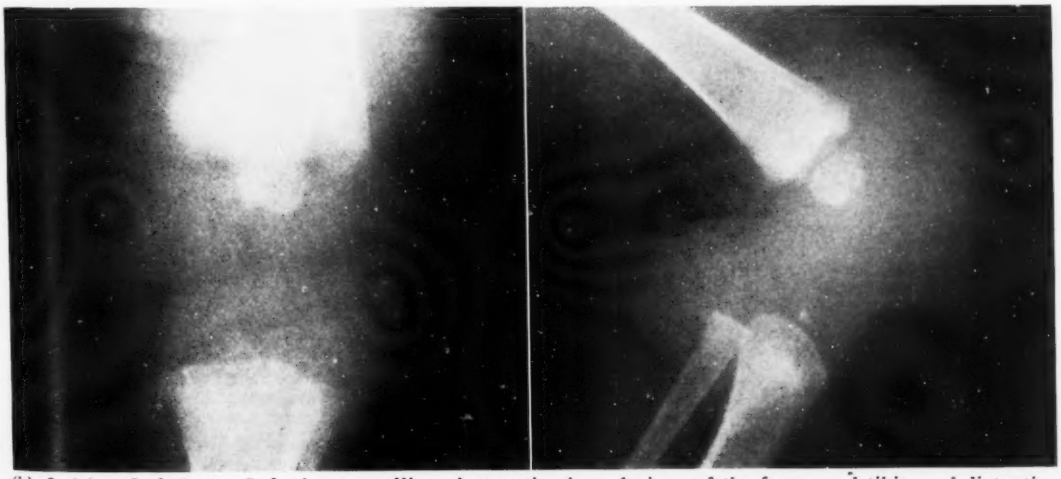
Osteoporosis of the bones of the joint was only detected early twice, and in each case in the hip. Later, it was seen in all the children.

Discussion

In the last decade the radiological diagnosis of liquid under pressure in the large joints of the body has been greatly advanced. Lewis[6,7] deserves much credit, as his approach to the study of soft tissues about the knee allows accurate recognition of capsule distention of free liquid. In a similar manner joint capsule distention has been radiologically defined in the hip by Drey[2], Hermel and Sklaroff[4]; in the elbow by Norell[8], Kohn[5], and Bledsoe and Izenstark[1]; and in the ankle by Weston[11]. Their contributions have been of great help and are the basis for the observations "fat mass displacement" in Table I, Section C. This was particularly true in the older children where the fat planes are larger, easier to see, and less frequently obliterated by local edema and inflammation.

Watkins et al[10] in 1956 stated "The roentgenographic examination may be of little help in the early stages of the disease, although occasionally the pressure of capsular distention may be revealed by the soft tissue shadows." Eyre-Brook[3] and Obletz[9] report much better experience in the hip; all the cases of Obletz[9] were diagnosed radiologically. Our review shows equal success with the hip, and similarly the other major joints of the extremities may be accurately assessed early in the disease.

We believe that the high incidence of positive and correct diagnosis in our series strongly supports the thesis that distention of the large joints in babies must be considered to be purulent until proven otherwise. We have not attempted to do a "control" study, because infants, particularly very young ones, with distention of large joints by fluid of non-infectious origin, are indeed rare. Exceptions must of course be made of clearly traumatic joint fluid collections, which are common at any age. If the radiologist will maintain a high index of suspicion regarding joint infection in early infancy, and search carefully for the cardinal signs of soft tissue swelling, bone separation and fat plane displacement in sick patients in this age group, diagnostic accuracy will increase. The importance to life and limb in such early diagnosis should be self-evident. In disease of



(b) & (c) — Left knee. Soft tissue swelling, destructive bone lesions of the femur and tibia, and distention of the capsule of the knee joint.

insidious onset and slow course (as in Case V above), every day counts; in fulminating cases, every hour wasted is disastrous.

Summary

A decade's experience of 35 infants and children with septic arthritis was reviewed. All had radiological signs of inflammation and nearly all had radiological signs of joint fluid in their first roentgenograms. Five case reports with illustrations present the clinical problem in the infant. The radiologist, when conscious of the nature of the disease, can be highly accurate in detecting those joints that likely contain pus.

Résumé

Il s'agit d'une revue de 35 cas d'arthrite septique chez des nourrissons et des enfants survenus au cours des dix dernières années. Tous les cas revus présentaient des signes radiologiques d'inflammation et presque tous avaient des signes radiologiques de liquide intra-articulaire dès les premières radiographies. Cinq cas rapportés avec illustrations servent à exposer le problème clinique chez le nourrisson. Le radiologiste, au courant de la nature de la maladie, peut identifier avec précision les articulations susceptibles de contenir du pus.

ACKNOWLEDGEMENTS: We wish to acknowledge gratefully the secretarial help of Miss M. J. Miles; Mr. J. Gunn and Miss J. Cox for help in preparing an exhibit; and Dr. H. T. Davenport, Dr. J. L. Shugar and Dr. B. St. J. Brown for reading the manuscript and giving valuable advice.

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BOOK REVIEWS

An Atlas of Normal Radiographic Anatomy, by Isadore Meschan with the assistance of R. M. F. Farrer-Meschan, 759 pages, 1446 illustrations on 412 figures. W. B. Saunders Company, Philadelphia, 1959.

Most of this excellent book consists of illustrations to define normal radiographic anatomy. This is achieved by a sketch of the method of positioning, an illustrative film, and a line drawing of the film with identification of the anatomical parts. All of the commonly used projections and many of the less frequently needed are included.

The atlas has been prepared for physicians who are not radiologists, resident radiologists and X-ray technicians. It meets their needs well as the emphasis is upon the lucid figures with sufficient text to unite them and orient the reader. The book need not be read completely but is easily used as a reference due to its simple and logical arrangement, and to a good index.

An earlier edition was the first book this reviewer read as a radiology resident, and it served well in rapid, yet accurate orientation. This newer, larger edition is a better book.

D.W.M.

Vertebral and Carotid Angiograms in Tentorial Herniations, by Hans F. Plaut, M.D., with a foreward by Juan M. Taveras, M.D., Charles C. Thomas — Publisher, Springfield, Illinois, 1961, $10.50.

The content of this book is the result of much basic research into the normal and abnormal anatomy of the tentorium cerebelli, especially the position and shape of the margins of the incisura and the relation of the posterior cerebral and superior cerebellar arteries to the incisura.

Part I contains many roentgenograms, diagrams and cadaver angiograms demonstrating these anatomical facts very clearly. Part II is a discussion of various cases of herniation through the incisura tentorii, both descending and ascending. Illustrations by means of angiograms (mainly vertebral) depict the various types of displacements of the posterior cerebral and superior cerebellar arteries. Side to side displacement of cerebral tissue through the opening in the falx cerebri is described and illustrated. The subject matter is easily understood and line drawings greatly enhance the reproductions of the roentgenograms.

The book has limited application because of the topics discussed. It does clarify many points about the diagnosis of herniation through the incisura tentorii and will be found very useful to those dealing with cerebral angiograms, even occasionally. The bibliography is complete.

134 pages (exclusive of bibliography).

133 roentgenographic reproductions and line drawing illustrations.

D.G.W.

CORONAL CLEFT VERTEBRAE and PERSISTENT NOTOCHORDAL DERIVATIVES OF INFANCY

* D. G. WOLLIN, M.D.
G. B. ELLIOTT, M.B., B.S.

A coronal cleft vertebra is one in which an axial radiolucent band is seen in the lateral roentgenogram of the spinal column, usually traversing partly or completely one or more adjacent vertebral bodies. This has been regarded, together with sagittal cleft vertebra, as an anatomical variant of ossification. We report three radiologically similar cases, one of which was due to persistence of axial notochord; one other was due to clefts of cartilage indenting the ossification centres of the vertebral bodies from each side. Notochordal remnants were present in the bodies of the vertebrae involved.

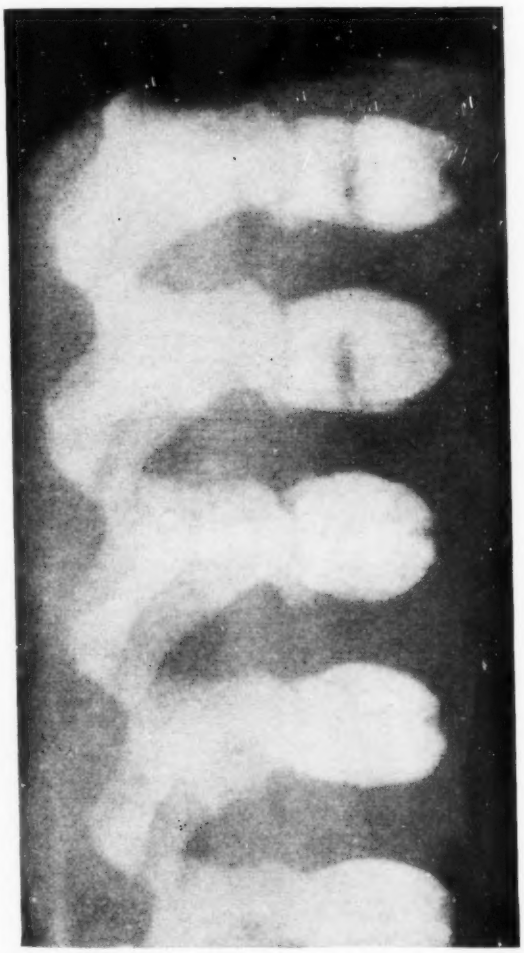

Figure I, Case 1.
Lateral roentgenogram of excised lumbar vertebrae. Radiolucent bands in bodies of L1 and L2 vertebrae correspond to persistent notochordal tissue.

Figure II, Case 1
Sagittal section of lumbar vertebrae. Persistent notochordal remnants corresponding to radiolucent defects in L1 and L2 vertebral bodies (white arrow). Small nodules of notochordal tissue in bodies of L3 and L4 not visible in roentgenogram (black arrows) (see Figure I).

Case 1. Baby Boy H.

This newborn male infant was examined within hours of birth because of persistent vomiting. Roentgenologic examination showed high intestinal obstruction, and on exploration volvulus of the mid gut with peritonitis was found. The infant died soon after surgical reduction.

Lateral views of the abdomen made during roentgenologic examination demonstrated coronal

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clefts in the vertebral bodies of L1 and L2. (Figure 1.) On section no coronal cleft was present, but a cylindrical core of gelatinous white cartilage was seen, running through each corresponding vertebral body (Figure 2). In line with these a tiny nodule of similar cartilage was seen buried in L3 and L4 vertebral bodies which had not been visible in the roentgenograms (Figure 2). The cartilaginous rods lay at the junction of the posterior one-third and anterior two-thirds of the antero-posterior diameter of these vertebrae. The affected bodies were slightly increased in antero-posterior diameter. Microscopically the section showed a central core of irregularly arranged cartilage cells in a very loose matrix of connective tissue mucin, indicating notochordal derivation. No columnar endochondral ossification was detected at the margins of the rod (Figure 3).

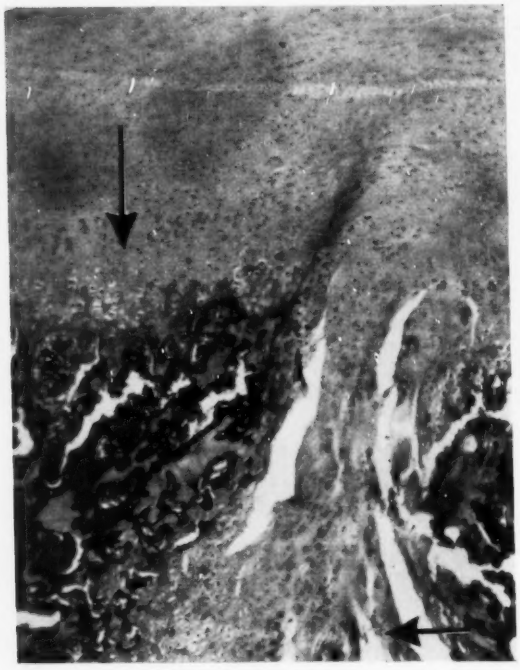

Figure III, Case 1
Photomicrograph of upper aspect of L1 vertebral body (see Figure II). Endochondral bone formation (long arrow) is seen on the disc surface. Axial core with notochordal features (small arrow), enters the vertebral body. This produced the radiolucent band visible in the roentgenogram.

Case 2. Baby Boy D.B.

A three week old male infant was admitted to hospital because of an upper respiratory tract infection. A lateral view of the chest fortunately included the entire spinal column. This showed a radiolucent coronal band in the body of L2 and a less well defined band in L4 at the junction of posterior one-third with anterior two-thirds of these vertebral bodies. The body of L2 was slightly increased in antero-posterior diameter, but decreased in transverse diameter (Figure 4). The child was discharged from hospital when the acute illness had subsided. No other anomalies were evident. Follow-up examination has not been possible.

In order to determine if this appearance was common in the pre-natal period, the vertebral columns of eight foetuses, born by spontaneous abortion and ranging in gestation from nineteen to twenty-six weeks were examined by means of lateral roentgenograms. One of these demonstrated coronal clefts, and it is described as Case 3.

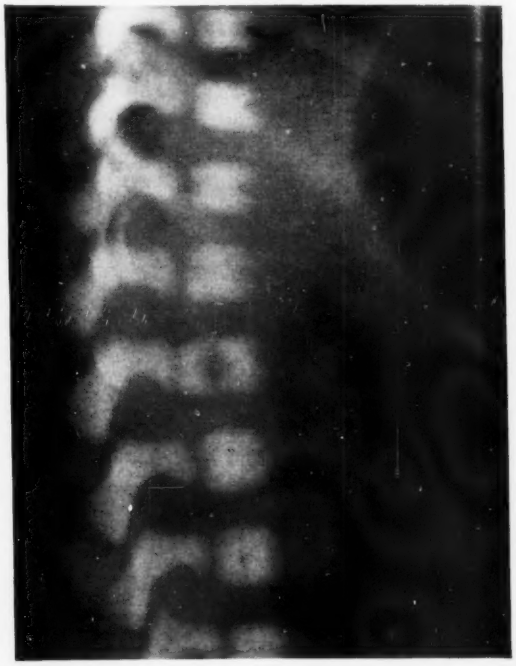

Figure IV, Case 2
Lateral roentgenogram of lumbar spine. An axial radiolucent band is seen in the bodies of L2 and L4 vertebrae, in the plane of the notochord.

Case 3

This was a five and one half month stillborn male foetus weighing thirteen and a half ounces. The mother, aged thirty-one, was healthy and had given birth to six apparently healthy normal children, all alive and well. The foetus showed a mild talipes equinovarus, but no other anomalies.

Roentgenograms demonstrated coronal clefts in the vertebral bodies of L1 and L2 (Figure 5). On serial section there was a bilateral coronal fissure in each of the two vertebrae, more marked on the right side (Figure 6). Microscopic examination showed normal endochondral ossification on each margin of the cartilage which filled these lateral clefts. However, the cartilaginous clefts showed no central fusion, a core of persistent notochordal cells filled the interval between them.

Discussion

Vertebral bodies are formed from fusion of two lateral centres of chondrification, in which a central solitary centre of ossification appears. Failure of fusion of lateral cartilaginous centres, with subsequent development of separate lateral ossification centres, results in a sagittal cleft vertebra. The appearance of a small posterior and a larger anterior

ossification centre results in coronal cleft vertebra. The cause of failure of fusion between chondrification centres is the crux in each case.

All authors are agreed that the situation for coronal defects is characteristically at the junction of the anterior two-thirds with the posterior one-third of the vertebral bodies. The defects have been described in this position in dorsal and lumbar vertebral bodies, but not elsewhere in the vertebral column. This corresponds to the position of the notochord. The notochord normally occupies a more anterior position in cervical and sacral levels, extending anteriorly even to the point of lying in contact with pharyngeal and pre-sacral soft tissues in some instances; hence the rarity of coronal defects in these segments of the spine. A number of authors viz. Reisner[7], Frets[5], Sereghy[9] and Schinz[10] have noted that the clefts are occupied by notochordal or "inter-vertebral disc tissue". Meyer-Burgdorff and Klose-Gerlich[6] show a horizontal cross section of a coronal cleft vertebra which is very similar to that illustrated in Figure 6. According to Schmorl and Junghanns[11] persistence of notochord plays a prominent part in disturbing vertebral development so that a "butterfly" vertebra results. In the cervical region Ehrenhaft[2], and Fallon, Gordon and Lendrum[3] have shown that notochordal persistence prevents fusion of laterally situated cartilaginous vertebral halves producing cervical sagittal cleft vertebrae. Cohen, Guido and Neuhauser[1] express the opinion that coronal clefts represent an anomalous method of ossification of vertebral bodies in which the ossification centres meet in the plane of the notochord. Such observations suggest that coronal and sagittal cleft anomalies of lumbo-thoracic vertebrae are a result of notochordal persistence inhibiting fusion of vertebral anlagen.

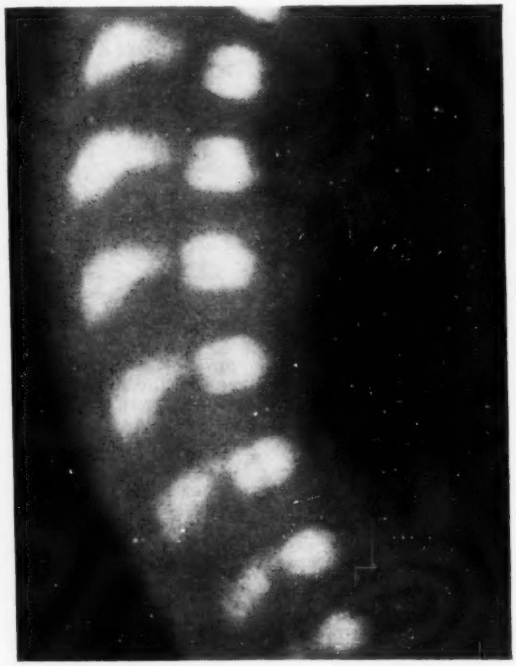

Figure V, Case 3
Lateral roentgenogram of foetal lumbar spine. Axial radiolucent band seen in bodies of L1 and L2 vertebrae.

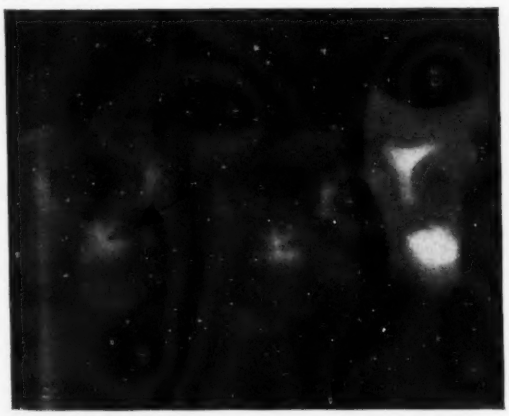

Figure VI, Case 3
Supero-inferior roentgenogram of L1 and L2 vertebral bodies. Coronal clefts demonstrated in bodies of L1 and L2 (arrow). Between the clefts in each centre of ossification a small nidus of notochord persists (not seen in above reproductions).

In one case reported the defect remained open until thirty-two months after birth at which time it was almost closed. There is a strikingly high sex incidence in favor of the male. Thus, of 28 cases reported by Rowley[8], 25 were in male infants. The three cases reported here were males. Associated congenital abnormalities are more frequent in foetuses and infants which show this anomalous ossification of the vertebrae. In Cohen's[1] series, out of a group of 200 infants being investigated for some type of congenital anomaly (tracheo-oesophageal fistula, imperforate anus, etc.), 13 had coronal clefts vertebrae, while in a control group of 200 picked at random, only 4 showed this condition. Some degree of meningocele occurred in 4 and, oddly enough, an imperforate anus was present in 6 of these 13 infants. One of the infants, in our report had a mid-gut volvulus, and another showed talipes equinovarus.

It is possible to detect this developmental aberration in utero, Fawcett[4]. Of Rowley's[8] 28 cases, 15 were demonstrated during pregnancy. In view of the possibility of associated congenital anomalies, this may be an important observation.

Summary

Three cases of "coronal cleft vertebrae" have been described, and in two, microscopic study was possible. One was due to a radiolucent axial rod of notochord, and in the other lateral clefts filled with cartilage were present in the plane of a notochordal remnant.

Observations from the literature indicate that notochordal persistence delays or prevents normal fusion of the usual vertebral anlagen, producing anomalies such as coronal and sagittal cleft vertebrae.

Generally, coronal cleft vertebra is considered as a variant without clinical implications, occurring almost exclusively in males, but reported series have shown a higher incidence of associated congenital anomalies. Its observation is important for these reasons.

Résumé

Les auteurs présentent trois cas dans lesquels le film de profil de la colonne lombaire démontre une bande radio-transparente dans un ou plusieurs des corps vertébraux à l'endroit où se trouve normalement la notochorde.

Ceci est considéré comme une variation normale de l'ossification des corps vertébraux lombaires ou dorsaux.

Deux faits intéressants ayant une corrélation réciproque sont mentionnés:

1 — cette variante s'observe presque uniquement chez les mâles.
2 — la coexistence d'anomalies congénitales est plus fréquente.

Cette anomalie peut par conséquent être considérée comme significative lorsqu'elle est découverte in utero.

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BOOK REVIEW

The Physics of Radiology, by Harold Elford Johns, M.A., Ph.D., F.R.S.C., LL.D. Charles C. Thomas, Publisher, Springfield, Illinois, 1961. 767 pages, $21.50.

The first edition entitled "The Physics of Radiation Therapy", published in 1953, has been completely revised and expanded to more than twice its original length. Besides enlarging and revising each of the original chapters, the author has added chapters on Physical Principles of Diagnostic Radiology, Clinical Uses of Isotopes, Radiation Protection, Radiobiology, Rotation Therapy and Combination of Fields for Clinical Uses.

The book discusses the entire field of radiation physics in a clear manner and provides the considerable data required for the correct application of radiation physics in both diagnostic and therapeutic radiological departments.

As in the first edition, the processes by which a beam of radiation imparts its energy to tissue is discussed in detail, particular emphasis being placed on energy absorption. By the use of many examples and problems, the author has clearly illustrated the concepts of absorbed dose and exposure dose. From this, the logic of using the rad as an expression of absorbed dose is more clearly understood.

The trend toward the use of cobalt, caesium and other supervoltage equipment by radiotherapists is reflected in the illustrative problems presented in the chapter on Combination of Fields for Clinical Uses.

An appendix crammed full of tables on absorption coefficients, conversion factors, stopping powers, depth dose data for circular, as well as rectangular fields, scatter functions, and selected isodose curves augments the many illustrated examples in the book. In addition, the author concludes each chapter with a set of problems.

The author has certainly achieved his goal of providing a most useful text book for both the student in physics as well as the student in diagnostic or therapeutic radiology — a must for all radiology departments.

S. Fedoruk
C. C. Burkell

BEAM FLATTENERS FOR A TELETHERAPY UNIT

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Atomic Energy of Canada Limited
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This article describes the performance of selective absorbers placed in the beam of a kilocurie teletherapy machine as a method to improve the shape of the radiation field from teletherapy units. A beam flattener attenuates the central portion of the emitted beam so that the distribution of energy is more nearly constant in this central region. The value of this straight line distribution lies in an increase to the useful beam width.

Equipment

The present work was carried out on a standard production unit of the Eldorado Super "G" Cobalt^{60} Teletherapy Machine manufactured by Atomic Energy of Canada Ltd. The unit was loaded with 3,200 curies of Cobalt^{60} in a 2 cm. diameter source having an output of 48 roentgens per minute at one metre from the bottom surface of the source. The adjustable diaphragm on the unit consists of 5.0 cm. thick tungsten blocks with vertical sides which are situated at 34.9 cm. from the source. The field measurements were made in a water bath phantom, having dimensions 76 cm. long, 51 cm. wide and filled to a depth of 44 cm., with an isodose plotting instrument capable of drawing the contours of constant dose rate that exist in the bath. The instrument consists of a waterproofed ionization chamber having a cavity 18.5 mm. long by 3.8 mm. diameter, which is attached to a servo-actuated remotely controlled arm, whose movements are duplicated by a similar mechanism driving a recording pen in a radiation free area. The plotter was built in the laboratories of A.E.C.L.

Construction

Early work showed the effect on the field of a given flattener varied both with diaphragm opening and with depth in the phantom. From the start, therefore, it was decided to design two flatteners to cover a representative range of clinical treatment conditions. The centre points of design chosen to meet these conditions were a phantom depth of 12 cm. with one field size at 20 x 20 cm.* and another at 14 x 14 cm. and both at 75 cm. source to skin distance. The flattener shape empirically evolved is one of spherical form with the two shapes differing only by their radii of curvature. The flatteners are fabricated from brass, and are only large enough to intercept the direct radiation emitted from the diaphragm port. Brass was chosen because it is readily available, is easily machined and, at short diaphragm to skin distances, the skin dose due to secondary electron emission is minimized[1]. In use the flatteners are placed in accessory guide rails attached to the bottom face of the therapy head; this enables them to be quickly and accurately positioned in the beam and to be easily removed when the optical beam is required in the preparation of a patient before treatment. Flattener "A" is 10.0 cm. square which is equal to the geometrical beam dimensions at the flattener position for a 20 x 20 cm. field at 75 cm. SSD. Figure 1, and flattener "B" is 7.0 cm. square equivalent to the geometric dimensions of the 14 x 14 cm. field at the position of the flattener.

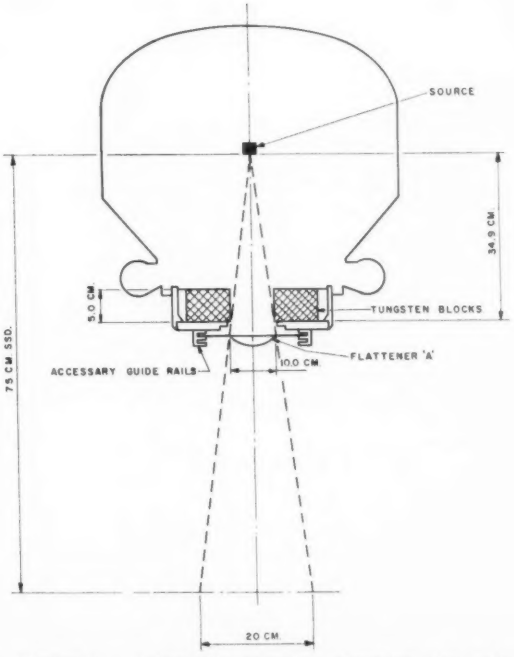

Figure 1 — Schematic drawing showing the relationship of the source, the tungsten defining block and the beam flattener relative to the geometrical beam.

* *All field sizes and diaphragm openings given here are expressed in terms of the geometrical field obtained from a point source.*

Beam Characteristics

In the isodose chart of Figure 2, where the normal and beam flattened curves have been drawn with no change made in the diaphragm aperture, with the treatment centre (TC) situated at a depth of 13 cm. and having a 90 per cent lateral dose uniformity, the beam width has been raised from 18.0 cm. to 21.2 cm., equivalent to an increase of 18 per cent. With flattener "B" and a 14 x 14 cm. field, the gain in beam width at 12 cm. depth is 1.4 cm., from 13.0 cm. to 14.4 cm., an increase of 11 per cent. If a 95 per cent lateral dose uniformity is required for a tumour at 11 cm. depth and a 20 x 20 cm. field, the flattened beam width is increased by 6 cm. over the normal curve, a gain of 43 per cent. Similarly with flattener "B" and a 14 x 14 cm. field the beam width increase is 3.5 cm., a 35 per cent gain. This quality of a flattener enables smaller diaphragm openings to be used for a given tumour dose uniformity, or conversely greater tumour dose uniformity for the same diaphragm opening. At depths less than that of the design point, the isodose contours become progressively overcompensated, and at greater depths progressively undercorrected. Figure 3 illustrates this.

A further effect of the beam flattener is in a reduced penumbra, where the penumbra width is arbitrarily defined as the distance between any designated pair of isodose lines measured at the depth of the 100 per cent line. The penumbra width between the 90 and 53.5 per cent lines for a 20 x 20 cm. field is reduced from 1.2 cm. for the normal curve to 0.8 cm. for the flattened curve, a reduction of 33 per cent, while the 53.5 to 15 per cent width remains unaltered. Using flattener "B" for the 14 x 14 cm. field the penumbra width is decreased by 18 per cent, from 1.1 cm. to 0.9 cm., between the 90 and 51 per cent isodose lines and is again unchanged between the 51 and 15 per cent lines.

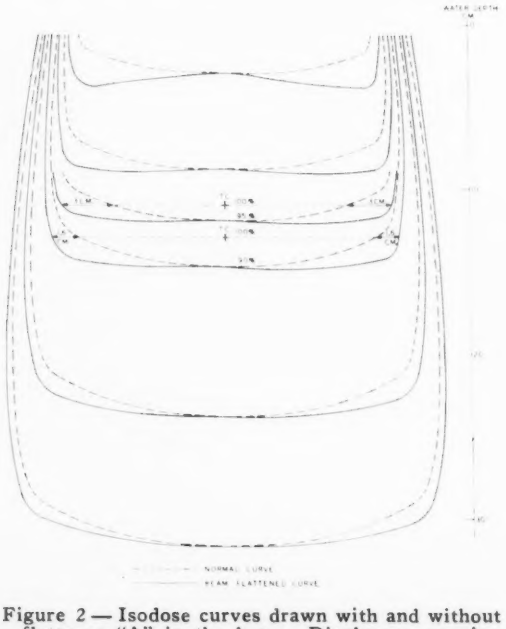

Figure 2 — Isodose curves drawn with and without flattener "A" in the beam. Diaphragm opening unchanged for the two sets of curves (20 x 20 cm at 75 cm SSD).

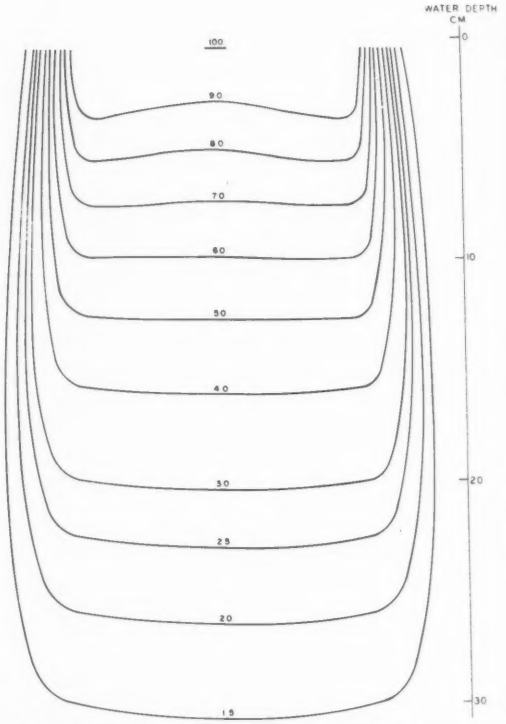

Figure 3 — Complete beam flattened isodose chart. Diaphragm opening set for a geometrical field of 14 x 14 cm at 75 cm SSD.

Although each flattener performs best at the field size and phantom depth selected, it is to be noted that their usefulness is by no means so narrowly restricted. The performance of flattener "A", as shown in Figure 4, at source to skin distances varying from 85 to 45 cm., all at a phantom depth of 12 cm., is clearly seen to be independent of the SSD. When smaller field sizes are employed predominance of the penumbra over the whole field grows larger and therefore the relative influence of the flattener over the central region grows less, cf. Figure 5. With the present two flatteners there is virtually no improvement at the 6 x 6 cm. and 4 x 4 cm. fields. Apart from the mid-section planes,

field characteristics were drawn through diagonal planes and at planes close to the edges of fields. The behaviour of the beam in rectangular fields as against the square field was also determined. Throughout these measurements the improvements noted earlier were maintained. Where extreme rectangular fields are encountered, namely 20 x 4 cm., the flattened beam becomes a little overcompensated.

The improvement in beam characteristics is obtained at some loss to the source output. Measured in air at 75 cm. from the source, flattener "A" causes a 29 per cent dose rate reduction in the centre of the field, and flattener "B" a 27 per cent reduction.

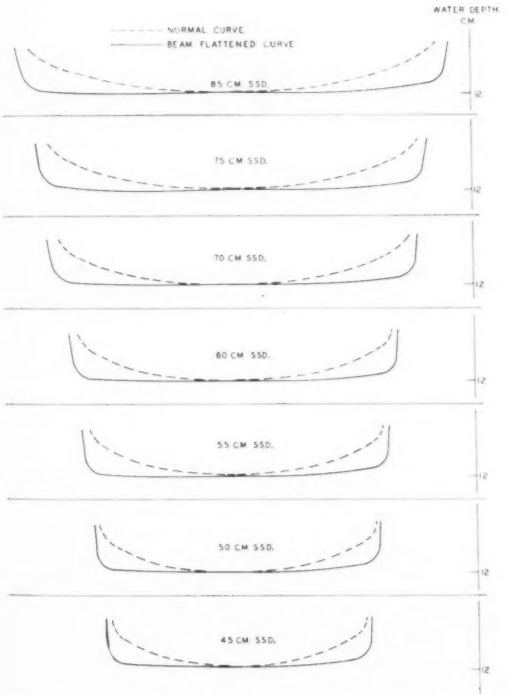

Figure 4 — Performance of beam flattener "A" at various source to skin distances. Diaphragm opening, equivalent to 20 x 20 cm at 75 cm SSD, kept constant.

Integral Dose Measurements

The ability of flatteners to increase the effective beam width should lead to a reduced integral dose in the patient. To measure this the diaphragm openings required to give equal tumour dose uniformity of 90 per cent (at a phantom depth of 12 cm.) were experimentally established for the normal and flattened beams (Figure 6). A Perspex bath measuring 29 x 30 x 30 cm. high was filled with ferrous sulphate dosimetric solution to

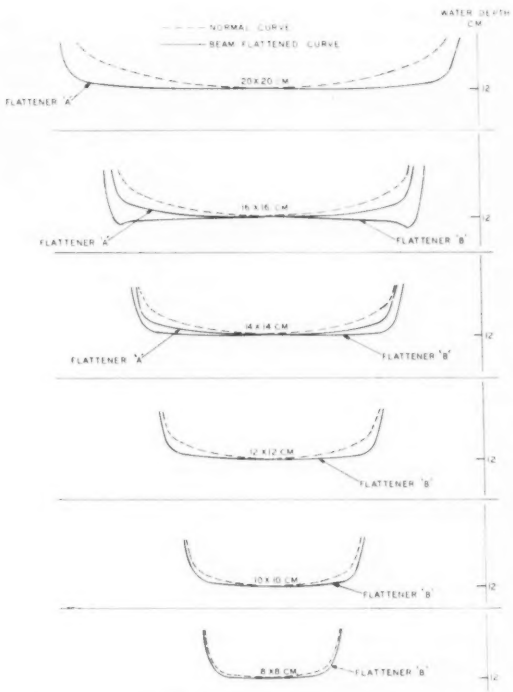

Figure 5 — Performance of the flatteners at various diaphragm openings with the source to skin distance held constant at 75 cm.

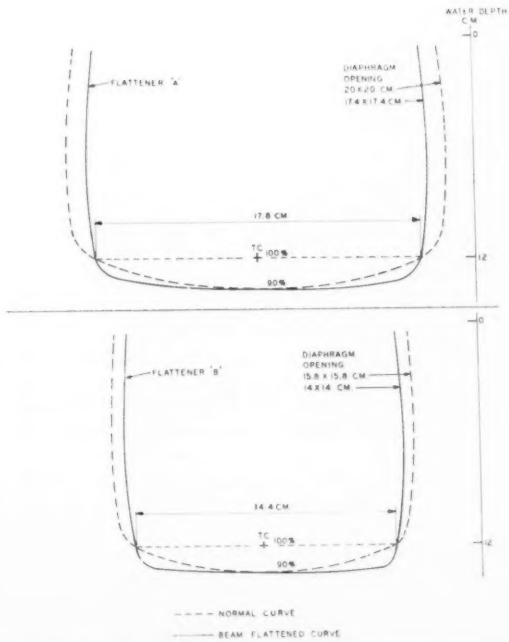

Figure 6 — Diaphragm openings for equal tumour dose uniformity for the integral dose measurements at 75 cm SSD.

a depth of 20 cm. and exposed in turn to the two different beams, while the central dose at 12 cm. depth was kept constant by adjusting the exposure time to compensate for the attenuation in the flatteners. At the conclusion of each exposure the solution in the bath was thoroughly stirred and a few millilitres of the homogenous liquid was then transferred to a Beckman spectrophotometer and the ultra-violet optical density measured. These particular readings could be reproduced to better than 1 per cent. The volume of solution in the bath was accurately determined, and held to this figure throughout the measurements, and from this known volume and the optical density reading the total absorbed dose, which was in the region of 4×10^9 ergs, could be ascertained. For a treatment width of 17.8 cm. with 90 per cent uniformity, flattener "A" reduced the integral absorbed dose by 10 ± 1.5 per cent while flattener "B", for a treatment width of 14.4 cm., reduced the integral dose by 3 ± 1.5 per cent.

Summary

Brass absorbers placed in the beam of a kilocurie cobalt unit have been shown to increase useful beam width, reduce penumbra width and to reduce the integral dose.

Résumé

Le faisceau de radiation émis par un appareil de télécobalt donne des courbes d'isodoses dont la partie centrale est sensiblement concave. Grâce à des disques de laiton de forme appropriée, il a été possible d'atténuer l'intensité centrale du faisceau et d'obtenir une courbe d'isodose pratiquement plane en son centre. Il s'ensuit une pénombre réduite avec un faisceau de diamètre agrandi. En utilisant un tel faisceau modifié on réduit le volume de tissu irradié et la dose intégrale sans pour cela diminuer la dose tumorale.

ACKNOWLEDGEMENTS: I wish to thank Mr. R. Kirkpatrick for undertaking the major part of the experimental work and to Mr. F. G. Rice for his continued interest and co-operation.

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BOOK REVIEWS

The Pathology of Ionizing Radiation, by Shields Warren. Published by Charles C. Thomas, 1961, $3.00.

This thirty-eight page booklet is a simplified introduction to the radiobiology of ionizing radiation. Although the text consists of an expanded lecture delivered before the Michigan Pathological Society, its stated purpose is to guide the informed layman between the "prophets of doom of the atomic age and the prophets' . . . . of nuclear utopia. Apart from examination of the excellent selection of references, many radiologists will prefer to wait for the author's presentation of a monograph describing the detailed pathology and radiobiology for which his studies during the past fifteen years have particularly qualified him.

R.M.C.

Radiologic Records, by Sister Christine Spirko, C.S.J., R.T., B.S., in R.T., M.A., F.A.S.T., Charles C. Thomas, Publisher, Springfield, Illinois, 304 pages, $8.50.

This book is, as stated in the forward by Dr Joseph Selman, an attempt by the author to do what no other has been fit to do — to collect, systematize and explain the various ways in which radiologic records may be managed. This reviewer's impression is that the result, while only a partial success, is nevertheless a valuable contribution.

The book is sufficiently small and handy to frighten no one who is trying to learn about this difficult and important subject. It is printed on high quality stock with clear, legible type, and moreover is readable. It contains much valuable information, obviously the result of years of study and well-organized, thoughtful reading. The bibliography is well arranged and clearly presented; it invites the reader to inquire further into particular aspects of the subject which interest or baffle him.

There are some outstanding weaknesses in this book, and it may be that these will be corrected in future editions. The photographic reproductions are on the whole of poor quality; consequently the chapter on "Medical Photography" is seriously weakened. A particular example of photography which will be noted by radiologists is the reproduction twice in the book (Figure 89 and Figure 100) of the same radiograph which is of indifferent quality. Some of the reproductions throughout the book also appear unnecessary, as for example, a photograph of a typewriter. Further instances of defects in editing are occasional sentences which are unnecessarily simple and therefore perhaps redundant in a book devoted to a specialized subject. One finds, for example on page 200, the statement "Radiographs are made with the use of heavy and expensive equipment called X-ray equipment."

All in all this compact and well-researched monograph will likely be most useful to those in search of information about some aspects of medical records on which they are as yet inadequately informed; the radiologist, technician or clerk can find in it a simple outline of basic principles and an excellent source of further reading. In this respect, the chapter on "Legal Aspects of Medical Records" is to be recommended particularly.

J.S.D.

EPIGLOTTITIS AND CROUP

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In the past few years a considerable number of publications have appeared, dealing separately with acute epiglottitis and with croup. No mention has been made, however, in any publication of the radiologic diagnosis of acute epiglottitis, and only one article[13] on the radiologic appearance of croup has appeared, reporting one case.†

Since both these diseases cause obstruction of the upper respiratory tract of infants and children, their recognition is of the greatest importance. The differential diagnosis between them is particularly important because, of the two, epiglottitis though much less common is more dangerous, with a rapidly progressive course and a high mortality rate, while croup is common, with a generally more benign course, though marked obstruction and death can occur.

In both conditions the clinical picture, though usually characteristic, can be difficult or confusing; in both, the onset may be so rapid that foreign body inhalation may be suspected, particularly if the history is incomplete or inaccurate.

The purpose of the present communication is to show that both acute epiglottitis and croup can be accurately diagnosed and differentiated by radiologic examination.

CLINICAL COURSE

Acute Epiglottitis This entity has also been called supraglottitis, supraglottic laryngitis, and acute epiglottic edema[6]. Its etiology, clinical course and pathology have been adequately described elsewhere[2,4,12,16] and will only be summarized here. The commonest, but by no means the only responsible infecting organism, is hemophilus influenzae. It occurs about equally in the two sexes, is commonest between the ages of 2 and 4 years, and rarely seen after the age of 8. The initial symptoms are often deceptively mild, including sore throat, fever and pain on swallowing. The further course is rapidly progressive; within a few hours severe pain, respiratory distress, inability to swallow, cyanosis, shock and death may occur. The characteristic posture of the patient with epiglottitis has been described as sitting or "tripoding" with chin thrust up, head hyperextended and tongue protruded with saliva pooling or drooling[5,23]. On examination of the pharynx, the red inflamed swollen epiglottis may be seen projecting from the hypopharynx, and is pathognomonic of the disease. There is some difference of opinion, however, about the safety of examination of such a patient with a tongue depressor[2,8,23]; most observers are agreed that the examination must be conducted gently and rapidly, that the tongue must be pulled forward rather than pushed back, and that repeated examinations are to be avoided[5]. The ease with which the inflamed epiglottis can be recognized is also somewhat controversial[12,23]. Pediatricians and otolaryngologists with experience in this disease have an obvious advantage in recognizing the abnormality in the hypopharynx from a brief, non-traumatic examination. The severely ill patient is, however, unable to swallow saliva, and the pooled secretions in the mouth and hypopharynx can make visualization difficult for even the experienced observer. Sudden death is common, and it is reported that permitting the child to be placed in the supine position may precipitate the terminal asphyxia. The cause of the asphyxia is not clear. It has been suggested that the swollen epiglottis is sucked into the larynx during inspiration, and that thereby a vicious circle is established, with the frantic attempts at inspiration making the obstruction more severe[12]. Our findings would suggest, however, that this phenomenon is likely not important; the aryepiglottic folds and epiglottis are so swollen and rigid that almost no difference in their position can be detected on films made in differing phases of respiration. We would therefore suspect that, contrary to the concept of the collapsing or mobile epiglottis, the rigidity of the supraglottic structures prevents normal swallowing of saliva. The swelling,

† *Jackson[10] in 1936 reproduced a lateral radiograph of the neck after tracheotomy "Showing obliteration of the lumen . . . by mucosal swelling complicating streptococcal laryngotracheobronchitis . . . " Hollinger[8] stated that "Roentgenograms of the larynx of an infant, particularly the lateral picture of the neck taken for soft tissues, are of great value. A careful roentgenogram of the larynx will demonstrate the true and false cords of the newborn infant, the epiglottis and the tracheal airway. Obstruction of the airway can frequently be detected by this means, establishing the diagnosis before the direct examination is made." He did not, however, publish any radiographic illustrations of this clear statement.*

which includes the false cords, narrows the airway to dangerous proportions; the oropharyngeal secretions cannot be swallowed, and may be aspirated into the narrow glottis, completing the respiratory obstruction.

The mortality from epiglottitis has been high in almost all reported series. Jones and Camps[12] in 1957 reported 26 out of 29 cases coming to coroner's post-mortem examination. Berenberg[2] in 1958 reviewed 42 cases of epiglottitis at The Children's Medical Center in Boston over a period of 8 years, and noted that 3 were dead on arrival and 2 died in hospital.

The experience at The Montreal Children's Hospital from 1951 to 1960 inclusive has been reviewed. The detailed report of the clinical findings and treatment will be the subject of another communication, but it can be said here that in this period 47 cases were admitted to hospital, with no mortality; 1 case was dead on admission.

Croup This term formerly referred to diphtheritic inflammation of the larynx and upper trachea. Non-diphteritic cases showing similar involvement were falled "false croup". The term croup no longer signifies diphtheria in the English language literature, but in the French-Canadian literature and that of continental Europe the term "false croup" is still used. It should be mentioned here that while diphtheritic croup is no longer seen in this part of Canada, we know of no way of distinguishing the radiologic findings of diphtheritic inflammation from that of croup caused by other agents. "Laryngotracheal bronchitis" and "acute laryngotracheobronchitis" are terms that appear to be used interchangeably for what is here called croup. The typical X-ray appearance referred to below indicates that the *swelling causing obstruction* is not so extensive as these longer terms suggest, and this plus the virtue of brevity has determined our preference for the name "croup"[10,13,19].

The inflammation, as suggested by the above-noted terms, is indeed extensive, as shown by many endoscopic and pathologic observations[1,3,8,11]. It involves the pharynx and hypopharynx and extends down to the smallest bronchi, and in some cases is associated with pneumonia. *The obstructive swelling*, which causes the acute symptoms and at times endangers life, is however localized to the subglottic trachea over a distance of about 1 to 1.5 cms. The explanation for the sharply localized subglottic swelling appears to be the loose attachment of the mucosa of the conus elasticus at this level[2,3,10,22]. Moreover, the swelling at this level must be entirely centripetal, at the expense of the lumen, since the rigid cricoid cartilage prevents lateral expansion of the soft tissues[8,14].

The etiology of croup is not well defined. While allergy and even a reaction to cold may be important in some cases, infection would appear to be the major cause. Viral infections may be at least as common or important as bacterial infections[4,22].

The age distribution differs from that of epiglottitis, though the two overlap; croup occurs in children in the first 2 to 3 years of life[3,10]. There is no sex predominance.

The course, while usually more gradual than that of epiglottitis, may be rapid and explosively sudden in onset, so that a foreign body obstruction may be simulated and in fact occasionally wrongly diagnosed. Signs of upper respiratory tract obstruction, including suprasternal and lower sternal indrawing, and dyspnea appear, as well as cough and hoarseness. The respiratory obstruction does not frequently become as severe as that of epiglottitis, and tracheotomy is only required occasionally. In spite of the rapid course and sometimes alarming signs and symptoms, the mortality is low.* Response to moisture and oxygen tends to be good, and recovery usually occurs within a few days. A tendency to recurrence is not uncommon, the same child having, over the first 2 or 3 years of life, several attacks of varying degrees of severity[18].

"Spasm" is often referred to in discussion of croup, but it is an ill-documented and probably little understood part of the disease[3,6,10,18]. The tendency for the child's symptoms to vary markedly from hour to hour and even from minute to minute and to respond dramatically to cool air and moisture have suggested that laryngeal spasm may contribute to the clinical picture. In addition, the correlation between the typical X-ray findings outlined below and the degree of clinical severity is not complete, and again the explanation for this may be laryngeal spasm. On the other hand, direct visualization of the spasm by endoscopy is infrequently reported, for the obvious reason that the

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* *Statements regarding low mortality in this paper refer to experience in recent years, when antibiotics and moisture have been extensively used in the treatment of croup. The careful review by Brenneman et al in 1938, of "a disease which carries a mortality of 50% or more" conveys a clear idea of the extreme gravity of the condition before the present era. In addition, it seems likely that the failure in the earlier literature to separate acute epiglottitis from the general group of "laryngotracheobronchitis" vitiated the statistics to some extent. Sinclair's report[25] in 1941 of ten cases of acute supraglottic inflammation caused by H. influenza Type B infection, though not the first description of the condition, was the beginning of present-day recognition of acute epiglottitis as a distinct entity.*

endoscopy is sometimes done under general anaesthesia and in any case as rapidly as possible, in order to demonstrate, and if necessary by-pass, the point of obstruction.

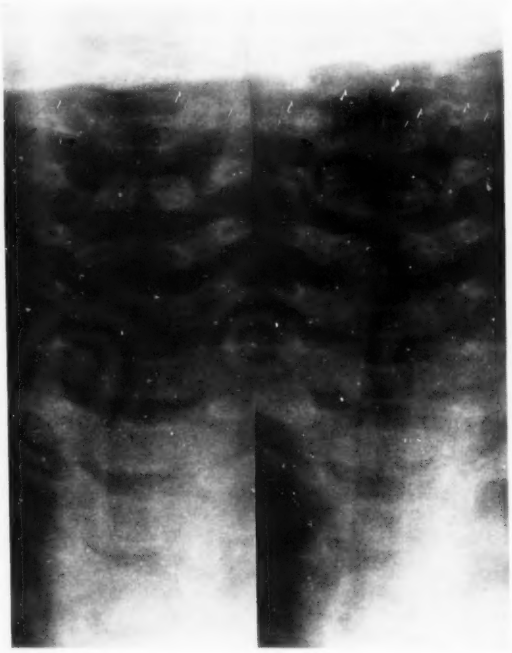

Figure 1(a) — Normal neck, at age 1 year, made during phonation. The true cords are almost touching. Note the air-filled piriform sinuses with tapered inferior tips at the level of the true cords.

Figure 1(b) — The same patient in quiet respiration. The vocal cords are widely separated. Tapering of the conus elasticus is just identifiable.

RADIOLOGIC FEATURES

Examination The only necessary X-ray examination to determine the cause of acute upper respiratory tract obstruction consists in AP and lateral projections of the neck in inspiration and expiration. It is frequently difficult or impossible to be sure that the films are exposed at the two extremes of the respiratory cycle, but two films in each projection in differing degrees of inspiration or expiration are, in our view, essential. There is such variation in the appearance of the normal and the abnormal upper respiratory tract during different phases of respiration and during phonation, that the two films in each projection are very valuable. If it is not clear from clinical examination whether the primary obstruction is in the upper or lower respiratory tract, films of the chest are also made, and depending on the combined clinical and radiologic findings, fluoroscopy of the chest and or neck may be necessary. It should be emphasised, however, that in the diagnosis of epiglottitis and of croup, the findings from simple inspiratory and expiratory films of the neck in AP and lateral projections are almost invariably pathognomonic.

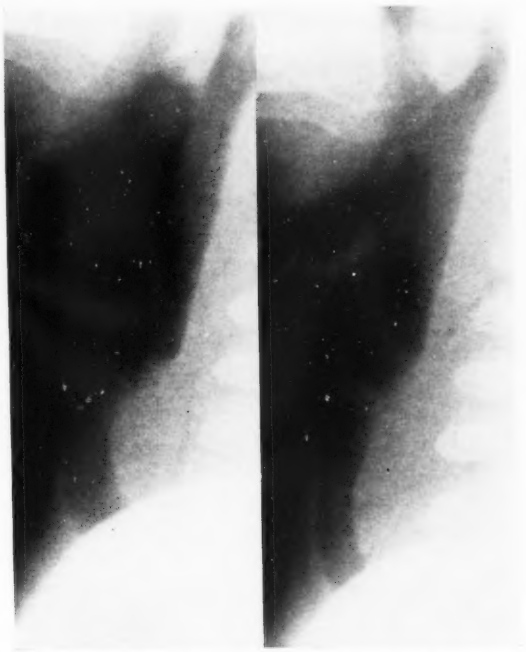

Figure 1(c) — Lateral film in inspiration. The pharynx and subglottic trachea are clearly defined and slightly dilated.

Figure 1(d) — Expiration. The pharynx and upper trachea show normal expiratory narrowing. The true and false cords, epiglottis and aryepiglottic folds are all sharply defined.

The infant is usually examined in the supine position, which permits the easiest immobilization of the patient and centering of the X-ray beam. If there is any suspicion that the child's condition is not as good in the supine as in the upright position, or if the child shows signs of "tripoding", or resists any effort to place him in the supine position, the films should be done upright, thereby avoiding the danger of sudden calamity in a case of epiglottitis.

Normal The structures of the upper respiratory tract and hypopharynx are easiest to demonstrate in the older child. In the infant they are less well defined but still readily recognizable. In the adult they are often partially obscured by calcified cartilage.

The subglottic trachea, often called the "conus elasticus"§, narrows in transverse diameter as the true (vocal) cords are approached. The contours of the lateral walls of the subglottic trachea, however, tend to be concave medially in the normal child. Moreover on inspiration and expiration the aperture between the vocal cords, and to some extent the width of the subglottic trachea, vary considerably, and this variation is an important sign of normality (Figures 1, 2).

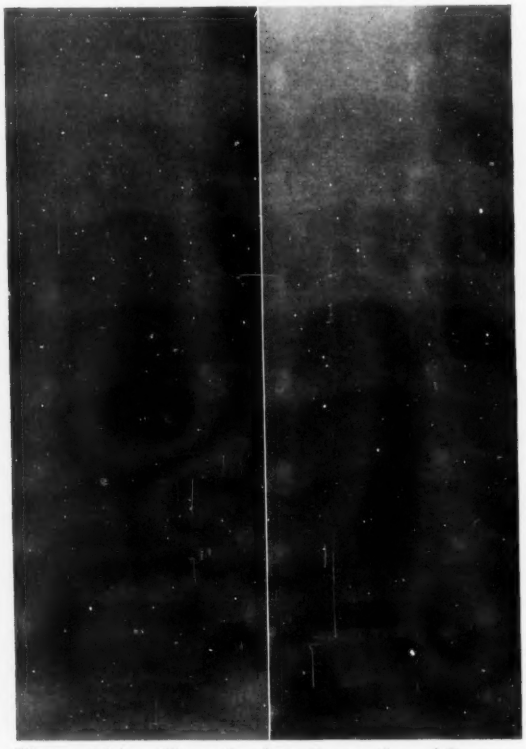

Figure 2(a) — Normal. Age 8 months. AP projection made during phonation. The true cords are almost fully apposed. Note the inferior tips of the piriform sinuses, which identify the true cord level.

Figure 2(b) — Phonation with true and false cords approximated. The piriform sinuses again indicate the level of the larynx, and show that this is normal laryngeal closure.

In the normal patient who is phonating, the vocal cords approximate to or touch each other, and this produces an apparent narrowing which may simulate tracheal stenosis. In this case the piriform sinuses, if filled with air, are visible on the AP films, and constitute a valuable landmark. Their tapered inferior ends correspond almost exactly to the level of the true cords, or at most 1 or 2 mms. superior to the cord level. Therefore the narrowing caused by the approximated cords can be recognized as normal even if the child is crying throughout the examination, provided that the piriform sinuses are visible on at least one film. Moreover it is rare to see the vocal cord narrowing exactly duplicated on two different films, whereas the narrowing produced by an obstructive lesion is much more constant.

Figure 3(a) — Normal. Age 4 months. Basal projection of the skull during phonation. The piriform sinuses are just visible.

Figure 3(b) — Less complete approximation of vocal cords during phonation. The piriform sinuses are not visible.

In the lateral projections the appearance of the subglottic trachea also varies slightly, depending on the width of the "conus elasticus" at the moment of the exposure. The negative contrast of the air-filled trachea diminishes slightly as the vocal cords are approached. Nevertheless the cords can almost invariably be seen on one or both films as sharply defined structures contrasting

§ *The term "conus elasticus" is used here, as it often is in practice and in the literature*[3]*, to refer to the portion of the airway immediately below the larynx, seen by radiologic or endoscopic methods, where the lateral walls taper medially to terminate in the vocal cords (Figures 1 and 2). Strictly speaking it refers to the cricothyroid membrane, whose thickened free upper border is the vocal ligament, or true (vocal) cord*[14].

clearly with the air-filled subglottic trachea. The laryngeal ventricle between true and false cords is frequently visible as well. The arytenoids, aryepiglottic folds, and epiglottis are normally clearly defined and readily recognizable. They vary in position, depending on respiration and phonation, but not in clarity of definition.

Occasionally, in milder, more chronic cases of upper respiratory disease, the basal projection of the skull is useful to demonstrate the anatomy of the hypopharynx, and particularly of the larynx (Figure 3).

Acute Epiglottitis The X-ray appearance of this entity is so characteristic and striking that it can hardly be confused with any other. The aryepiglottic folds and epiglottis are massively swollen, and appear to fill the entire hypopharynx. The swelling stops abruptly and dramatically at the larynx, the subglottic trachea being clear[16]. There is frequently slight widening of the retropharyngeal or pretracheal soft tissues, but the problem of differentiation from acute retropharyngeal inflammation or abscess virtually never arises.

As mentioned above, the appearance of acute epiglottitis on two or more lateral films of the neck of the same patient shows almost no variation, indicating extreme rigidity of these structures caused by the inflammatory swelling. Thus, two films of the patient can be superimposed on each other almost exactly, and the appearance is so typical that the films of one patient can be almost superimposed on those of another.

The only problem that is likely to be encountered in recognition of this entity by radiologic methods is that an observer unaware of the appearance may not recognize it as being abnormal; this likely accounts for the occasional reference in the literature to cases of epiglottitis in which "X-rays of the neck and chest were clear"[12]. Such examinations have usually been made to exclude a foreign body, and not to assess the structures of the upper respiratory tract[17].

We have never observed the supraglottic structures fluoroscopically in this condition, for the obvious reason that the X-ray examination should be kept to a minimum, and examination in a darkened room should be avoided if possible.

The typical radiologic and clinical picture of acute epiglottitis has also been encountered by us in children who have sustained burns of the pharynx during accidental ingestion of very hot water. These cases we intend to report in a separate communication.

Croup The radiologic findings in croup[13] are more variable than are those of epiglottitis, and at times more difficult to recognize, since all degrees of severity can be encountered. In a typical case, however, they are quite distinctive, consisting of a localized swelling of the subglottic trachea for a distance of approximately 1 to 1.5 cms. The "conus elasticus" as seen in AP projection shows medial convexity of its lateral walls, instead of the medial concavity of the normal patient. There is some variation in the degree of this tracheal airway narrowing, depending on the phase of respiration and on whether the patient is phonating, and there is some variation in the ease with which the abnormality can be visualized on AP as compared with lateral projections. For this reason both AP and lateral films in inspiration and in expiration are particularly valuable in recognizing or excluding this abnormality.

Above the level of the true cords the structures are normal. Slight thickening of arytenoid and aryepiglottic folds is sometimes seen but not to the degree where it could reasonably be confused with acute epiglottitis.

A phenomenon which may cause confusion in croup is secondary collapse of the trachea. This is not infrequently seen, and invariably occurs during inspiration. It will therefore simulate a diffuse narrowing of the trachea on the inspiration films, but the narrowing of all but the subglottic trachea will disappear during expiration (Figures 6, 7). Whether this secondary tracheal collapse is of importance in aggravating the signs and symptoms of the disease is not as yet clear. It must be distinguished from primary tracheomalacia of infancy, which is a tracheal narrowing in AP diameter, caused mostly by invagination of the pars membranacea of the trachea, and which occurs characteristically and only on expiration. Above the larynx, the hypopharynx is often unusually or even abnormally dilated in inspiration; this is due to the inspiratory pressure gradient across the site of obstruction during the violent inspiratory efforts (Figure 7).

The sharply localized subglottic swelling as seen by X-ray explains why the child with severe croup is immediately relieved by tracheotomy, as is the child with epiglottitis.

Fortunately, the more severe the abnormality, the more characteristic and easily recognizable it becomes on the radiographs (Figures 6, 7).

Occasionally, infants in the first year of life have a more chronic form of croup, whose etiology is not clear. This causes mild but definite symptoms. The radiologic findings

too are mild but definite, and their recognition is of value in establishing the extent, severity and location of the disease (Figure 9). We have seen two such cases in early life, both of whom have responded over a period of several weeks to conservative therapy.

The necessity for X-ray examination The experience with these diseases in our institution and in the published literature unequivocally indicates the need for early diagnosis and treatment[2,3,6,12]. It should be equally clear that not all cases of acute upper respiratory tract inflammatory disease of infancy and early childhood require radiologic examination. If the diagnosis can be made with reasonable certainty from clinical examination, it is evident that treatment must be undertaken immediately, and that X-ray examination, is unnecessary and in fact may cause undue delay in the initiation of treatment. On the other hand, even in the expert hands of otolaryngologists and pediatricians familiar with these conditions, the clinical picture can sometimes be difficult or confusing. Moreover, not all such cases are initially seen by specialists thoroughly familiar with the condition. For example, of the 42 cases of epiglottitis reported by Berenberg and Kevy[2], only one had been referred to the hospital with the correct diagnosis. X-ray examination can sometimes be of critical value in establishing the correct diagnosis when it is in doubt. We have also been impressed by the negative value of radiologic findings. In children presenting with signs and symptoms suspicious of upper respiratory tract obstruction, negative findings on radiologic examination can sometimes be of great value, and are highly reliable.

Since epiglottitis, though uncommon, is not rare and is very dangerous, and since croup can be dangerous and is common, the radiologist should become familiar with the normal and abnormal appearance of the upper respiratory tract in infancy and childhood. This is an area which he has up to now almost completely neglected.

Case I: T. G., Male, Age 4

This patient had been in good health until the day of admission to hospital. Ten hours before admission, he woke in the morning with a "croupy cough". He soon began to complain of sore throat and was found to have a fever of about 101° F. He stayed in bed during the morning, with no improvement. Early in the afternoon he developed difficulty in breathing with hoarseness of voice. He refused drinks and was unable to swallow fluids. There was no cyanosis. He was seen late in the afternoon by his pediatrician who advised immediate hospitalization.

On admission he was a normally developed and nourished white male in acute respiratory distress. In addition to marked dysphonia he showed tachypnea, subcostal indrawing, and marked supraclavicular indrawing. Examination of the chest showed no other abnormality. His temperature was 100° F, pulse 138 per minute, respirations 34 per minute. During the examination of the pharynx, on admission, he suddenly stopped breathing. An anaesthetist was called, an intubation done immediately, and respiration was rapidly reinstituted. He was unconscious however for about half an hour. Immediately after the intubation, a tracheotomy was done by the otolaryngologist (Dr. J. Baxter). Visualization of the pharynx and hypopharynx at the time of the intubation showed the typical findings of acute epiglottitis, with red and markedly swollen epiglottis. Following the tracheotomy he had a stormy course for 3 to 4 days. Cellulitis developed on the right side of the neck, but responded to conservative treatment. Full recovery of consciousness and alertness did not occur for almost 3 days. The tracheotomy tube was removed without difficulty on the sixth day. and he was discharged as recovered on the eighth day. Laboratory findings on admission were non-contributary. The radiologic examination of the neck done at the time of admission (Figure 4) showed massive swelling of the epiglottis and aryepiglottic folds. The bacteriologist's report on a throat swab done on admission was "mixed throat flora, from which no accepted pathogens were isolated". No culture specimen was obtained at the time of tracheotomy.

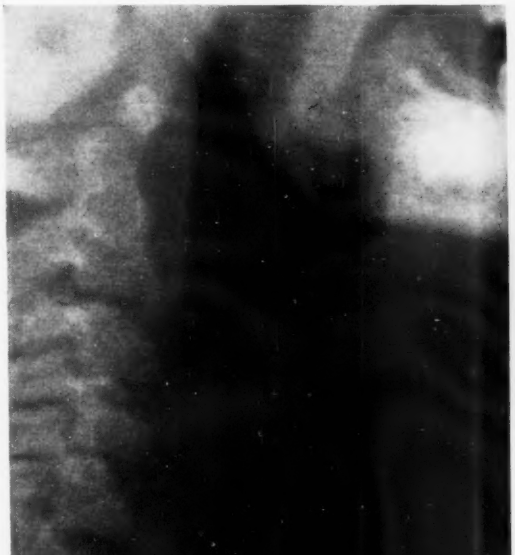

Figure 4 — Case I. Acute epiglottitis. Age 4 years. A lateral projection of upper neck and pharynx, showing the typical picture of swollen aryepiglottic folds and epiglottis. The upper trachea is clear.

Case II: C. I.

A four and a half year old female who was well until two days before admission, began to complain of a sore throat during the evening. On the following day, dysphagia was noted and no fluid could be ingested. On the day of admission she was seen in the morning at home, and noted to have dysphagia and stridor, with low grade fever, diffuse reddening of the pharynx, slight croupy inspiration, tender large cervical lymphnodes and scattered inspiratory rhonchi. She was then given 300,000 units of procaine penicillin, and visited again at home at 6 p.m. At that time, because of increasing toxicity and stridor and failure to take any fluids by mouth, with resultant dehydration, admission to hospital was recommended. Until the time of admission, no dyspnea, chest wall retraction, or cyanosis had been present. It was noted however that she was "spitting a great deal of frothy material".

Figure 5(a) — Case II. Acute epiglottitis. Age 3 years. Lateral film of the neck showing massive supraglottic swelling.

On admission at about 8 p.m. she appeared to be a well-developed child in fairly acute respiratory distress, and moderately dehydrated. There was an audible inspiratory stridor, and the mouth was kept open. The voice was hoarse, but she was not coughing. The respiratory rate was 22 per minute, the temperature 100° F, and the pulse 136 per minute. There was slight suprasternal indrawing and no substernal indrawing. The chest was hyperresonant, the breath sounds harsh, and loud rhonchi and rales were audible on both sides. The epiglottis was not visualized on admission. X-ray examination immediately following admission showed typical changes of epiglottitis (Figure 5a).

She was put in a croupette, given antibiotics by intramuscular injection, fluids and oxygen. In spite of this conservative therapy the child's condition worsened, as shown by increasing toxicity, restlessness and rising pulse rate (to 140 per minute). Examination by the otolaryngologist (Dr. A. Cohen) at 11 p.m. showed marked redness and swelling of the epiglottis. Tracheotomy was decided on immediately and carried out at 11:45 p.m., about 4 hours after admission. Post-operatively the child did well. For 3 days there was a low-grade fever of about 100° F, with diminishing epiglottic swelling and redness. One week after admission the tracheotomy tube was removed without incident, and 10 days after admission she was discharged as cured. The bacteriologic report (from laryngeal culture at the time of operation) was that normal flora was found (possibly because of penicillin given prior to admission). A lateral film of the neck on the day after tracheotomy showed that the arvepiglottic swelling had already diminished appreciably (Figure 5b).

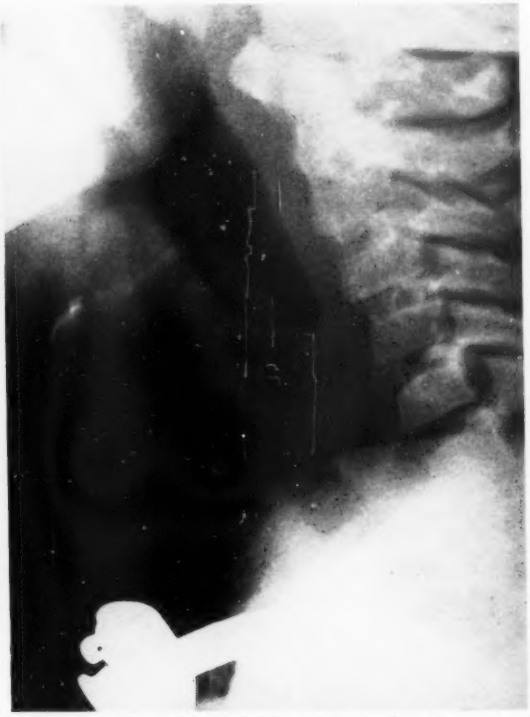

Figure 5(b) — Lateral film of neck showing recovery occurring, one day following tracheotomy.

Case III: R. B.

This 14 month old child was perfectly well until the onset of typical acute coryza one week before admission. The evening before admission he had a brief period of inspiratory stridor which disappeared spontaneously. About 3 hours before admission the stridor appeared again, accompanied by moderate respiratory distress. On admission to hospital the same evening, he was a well-developed, well-nourished child with marked inspiratory stridor, croupy cough, slight tachypnea, marked suprasternal and supraclavicular as well as intercostal indrawing. The temperature was 100.2° F, respirations and pulse were 30 and 128 per minute repectively. X-ray examination on admission showed (Figure 6) severe subglottic swelling of the trachea, which narrowed its lumen to a slit, and was typical of croup.

Following admission the patient was placed in a croupette with oxygen and high humidity. He was given sulfonamide by mouth and penicillin intramuscularly. He improved rapidly, and was removed from the croupette 2 days after admission, and discharged as cured 4 days after admission.

Case IV: B. B.

This 3 year old female was perfectly well until approximately 24 hours prior to admission, when she developed a croupy cough. During the 24 hours before admission the cough progressed in severity, and respiratory distress appeared and became more severe. There was no fever and no loss in appetite. About 4 hours before admission she was given penicillin intramuscularly.

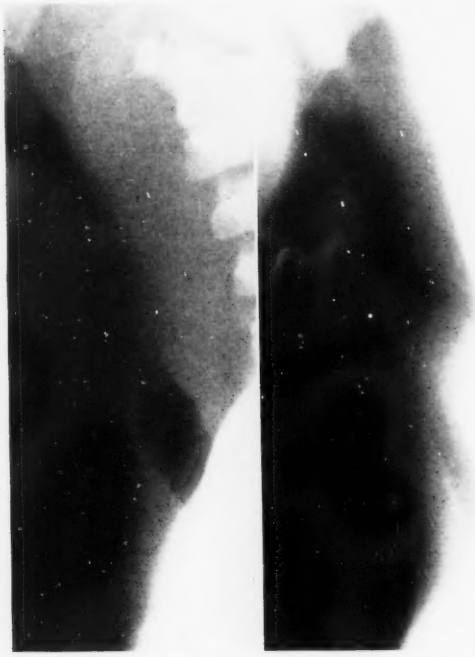

Figure 6(a) — Case III. Croup. Age 1 year. Lateral projection of the neck, showing severe and sharply localized subglottic swelling, with normal expiratory pharyngeal collapse above, and normal trachea below.

Figure 6(b) — Same patient on inspiration. Subglottic swelling is still present, but much less obvious. The pharynx is dilated; the lower trachea is clear, but shows some secondary narrowing.

On admission physical examination showed a well-developed child in obvious respiratory distress with a croupy cough, moderate suprasternal indrawing and occasional intercostal indrawing. The pulse was 150 per minute, respirations 42 per minute and temperature 100.4° F. Examination of the chest revealed no significant abnormality except tachypnea and tachycardia. Examination of the pharynx showed it to be red and injected; the epiglottis was visualized and appeared normal. Radiologic examination of the neck on admission (Figure 7) showed mild but definite subglottic swelling, typical of croup.

Following admission she was placed in a croupette with oxygen and high humidity. Tetracyclin was given intramuscularly. Within 24 hours her condition was markedly improved. No complications occurred and she was discharged as improved 3 days after admission. Bacteriologic examination of the throat swab was reported as showing mixed throat flora from which no accepted pathogens were isolated.

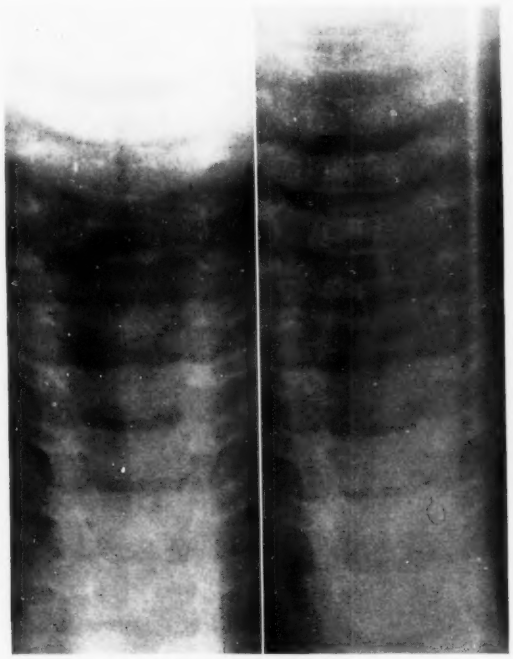

Figure 6(c) — Same patient in AP projection. Subglottic swelling is severe, with the lateral walls of the conus elasticus convex medially. The tip of the piriform sinuses are just visible, indicating the level of the larynx at the upper end of the obstruction.

Figure 6(d) — Expiratory film shows almost no difference in obstructive swelling. The piriform sinuses are not visible.

Case V: J. B.

A 6 year old male child who had a cold for several days. Shortly before visiting the hospital, he suddenly developed croupy cough and dyspnea. He was brought to the emergency department by his parents. Indrawing of the thoracic wall on inspiration was noted, together with some dyspnea, but no stridor. The pharynx was mildly inflamed. The epiglottis was not visualized. X-ray examination of the neck (Figure 8) showed moderate and obvious subglottic swelling of the trachea, with normal supraglottic structures. He was not admitted to hospital, though admission was seriously considered. He was treated at home with sulphonamides and penicillin, and made an uneventful recovery within 2 to 3 days. The parents reported recently that in the nine months since this acute illness he has been entirely well.

Case VI: M.D.

This 5 week old infant was well until 3 days before admission to hospital when he developed a cold, cough and fever. The parents noted some difficulty in breathing on the day of admission, and called their pediatrician (Dr. B. Quesnel), who visited the child at home, and found typical signs of croup, with dyspnea, sternal and intercostal indrawing, but no cyanosis. By the time he had arrived at the hospital, improvement had occurred.

Figure 7(a) — Case IV. Croup. Age 2 years. Lateral projection in inspiration showing subglottic swelling. The pharynx is dilated and the trachea below the obstruction is collapsed during marked inspiratory effort.

Figure 7(b) — Same patient in partial expiration. The pharynx is partially collapsed; the trachea below the obstruction is normal in calibre. The obstruction in the subglottic trachea is not as obvious as during inspiration.

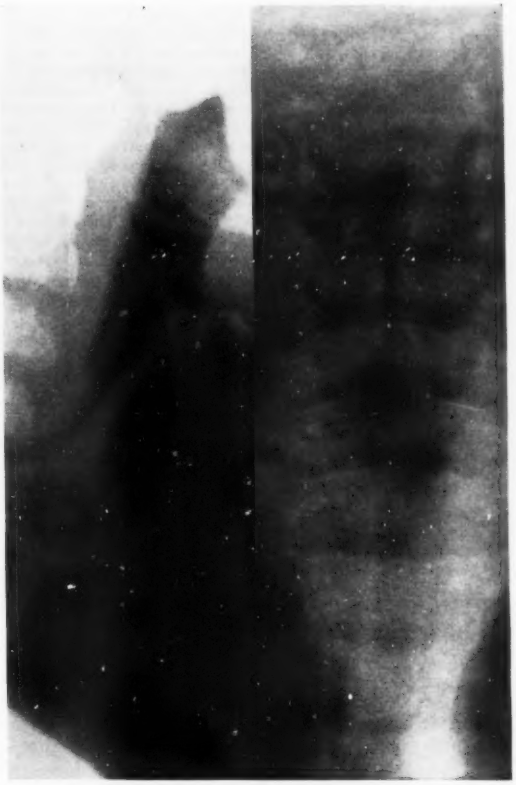

Figure 8(a) — Case V. Croup. Age 6 years. Lateral projection showing typical and sharply localized subglottic obstruction of moderate degree.

Figure 8(b) — AP projection showing markedly convex subglottic mucosal swelling. The piriform sinuses are narrower than average, but visible.

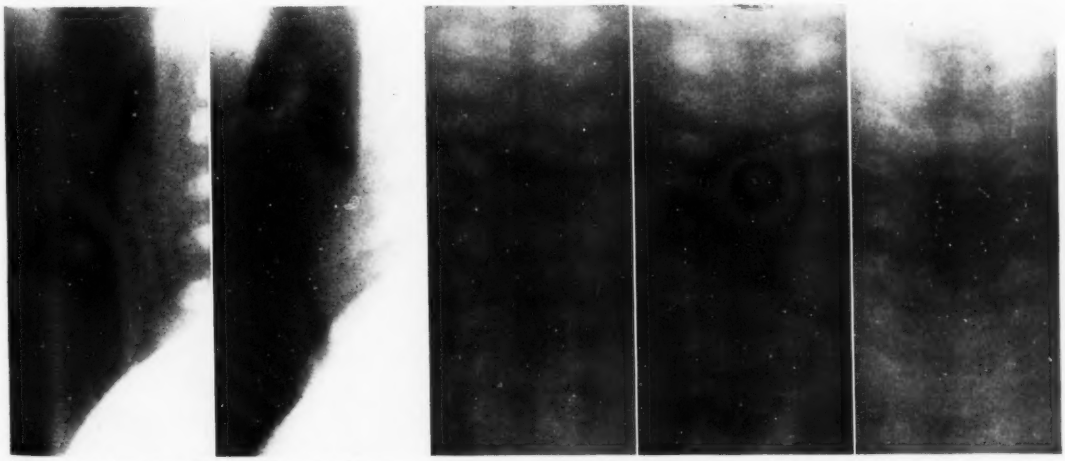

Figure 9(a) — Case VI. "Chronic" croup. Age 6 weeks. Lateral film during expiration showing typical subglottic swelling.

Figure 9(b)—Lateral film during inspiration showing moderate secondary lower tracheal collapse.

Figure 9(c) — AP projection during phonation showing subglottic swelling and approximation of the vocal cords.

Figure 9(d) — AP projection during quiet respiration showing subglottic swelling, and narrowing of the airway. Complete recovery occurred gradually over the next month.

Figure 9(e) — AP projection during quiet respiration one month later shows that the subglottic swelling has almost completely subsided.

Physical examination on admission showed a well-developed, well-nourished white infant in no acute distress, with inspiratory stridor but no chest wall retraction. There were some coarse, transmitted rales on both sides of the chest. The temperature was 98° F, pulse 135 per minute, and respirations 28 per minute. The day after admission, moderate to severe respiratory distress appeared, and the respiration and pulse increased to 60 and 154 per minute respectively. Sternal and substernal intercostal indrawing were marked. The mouth showed thrush and the throat was infected. Inspiratory stridor was present.

Films of the neck on the day of admission showed subglottic narrowing of the trachea (Figure 9) of moderate degree.

When this increase in signs and symptoms occurred, the patient was placed in an incubator with oxygen and high humidity for 4 days, and was given antibiotics. He began to improve on the third day of admission, but improvement was gradual. Six days after admission his condition was considered to be very good, and films of the neck showed that the subglottic swelling was still present, and mild to moderate in degree. He was discharged 7 days after admission, but continued to have noisy breathing at home for several weeks. The final X-ray examination was done approximately 1 month after admission. It showed almost complete disappearance of the subglottic swelling (Figure 9e), and the mother reported that although the breathing was still noisy it was almost back to normal. Within the next month his recovery was complete. as reported by Dr. Quesnel on follow-up examination.

Summary

Epiglottitis and croup are upper respiratory tract inflammations, causing obstruction in infants and young children. Though similar in many respects, they are strikingly and importantly dissimilar in prognosis. Epiglottitis, though less common, has a high mortality with a tendency to sudden death, while in croup the course, though rapid, is less often fatal. Both of these diseases can be accurately diagnosed by X-ray examination of the neck, using inspiration and expiration films in AP and lateral projections.

Résumé

L'épiglottite et le croup sont des processus inflammatoires produisant des obstructions des voies respiratoires supérieures chez les nourrissons et les jeunes enfants. Bien que ces entités se ressemblent sous plusieurs aspects, il est de la plus haute importance de les distinguer en vue de leur pronostic respectif. L'épiglottite, certes moins fréquente, a un taux élevé de mortalité surtout par mort subite, tandis que dans le croup le cours de la maladie est moins rapide et moins souvent fatal. Dans les deux cas un diagnostic radiologique précis peut être établi à l'aide de clichés du cou de face et de profil, en inspiration et en expiration.

ACKNOWLEDGEMENTS: The valuable help and co-operation of Dr. James Baxter of the Department of Otolaryngology, Dr. D. W. MacEwan and Dr. B. St. J. Brown, Miss M. J. Miles, and the Technical Staff of the Department of Radiology, The Montreal Children's Hospital, is gratefully acknowledged.

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FETAL CERVICAL HYPEREXTENSION

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The presence of fetal cervical hyperextension may be of serious import. The purpose of this paper is to discuss fetal cervical hyperextension as an indication of fetal morbidity and as indication for caesarian section. Also presented is a report of an unusual case of fetal cervical hyperextension associated with abnormal findings in the cervical cord.

Case Report

A radiograph of the mother's abdomen taken before the birth of the infant showed a breech presentation and an abnormal attitude in the form of hyperextension of the neck, such that the occiput appeared to touch the back of the fetus. (Figure 1).

The baby's delivery was uneventful; however, the infant breathed poorly and showed poor movements of his arms and legs. There was a total paralysis of the intercostal muscles. Breathing was described as being entirely diaphragmatic and this was confirmed by fluoroscopy. The Moro mass reflex was markedly diminished; the stepping reflex was present. The facia , sternomastoid and trapezius muscles seemed to be unaffected. The reflexes of the upper limbs were present and equal, whereas those in the lower limbs were absent.

A lumbar puncture at the age of two weeks revealed a slightly yellowish cerebrospinal fluid. The laboratory findings were: red blood cells — 63 per cmm., white blood cells — nil, protein 1000 mg. %, chlorides — 701 mg. %, sugar — 54 mg.% and also a marked increase in globulin.

Radiographs of the cervical spine taken at the age of four weeks were normal. A myelogram at the age of five weeks via the cisternal route showed a complete block at the level of the seventh cervical vertebra. The subarachnoid space superior to the block was widened at the expense of the cord. (Figure 2.)

At the age of six weeks an exploratory laminectomy was done. This revealed narrowing of the dural canal at the C5 to T1 level. On opening the dura, obliteration of the subarachnoid space was demonstrated and normal cord was not identifiable. No corrective surgery was attempted. The child developed bronchopneumonia and died two days following operation.

At postmortem the immediate cause of death was shown to be bronchopneumonia. The cervical cord at the level of C5 to T1 was narrowed to a width of 2 mm. for a distance of 1.5 cm. Microscopic section showed this area of the cord to be replaced by glial and fibrous tissue. No other abnormality of the nervous system was found.

Discussion

This case of fetal cervical hyperextension demonstrated narrowing of the dural canal, obliteration of the subarachnoid space with spinal fluid block, and gliosis of the cervical cord. It is not certain whether the lesion

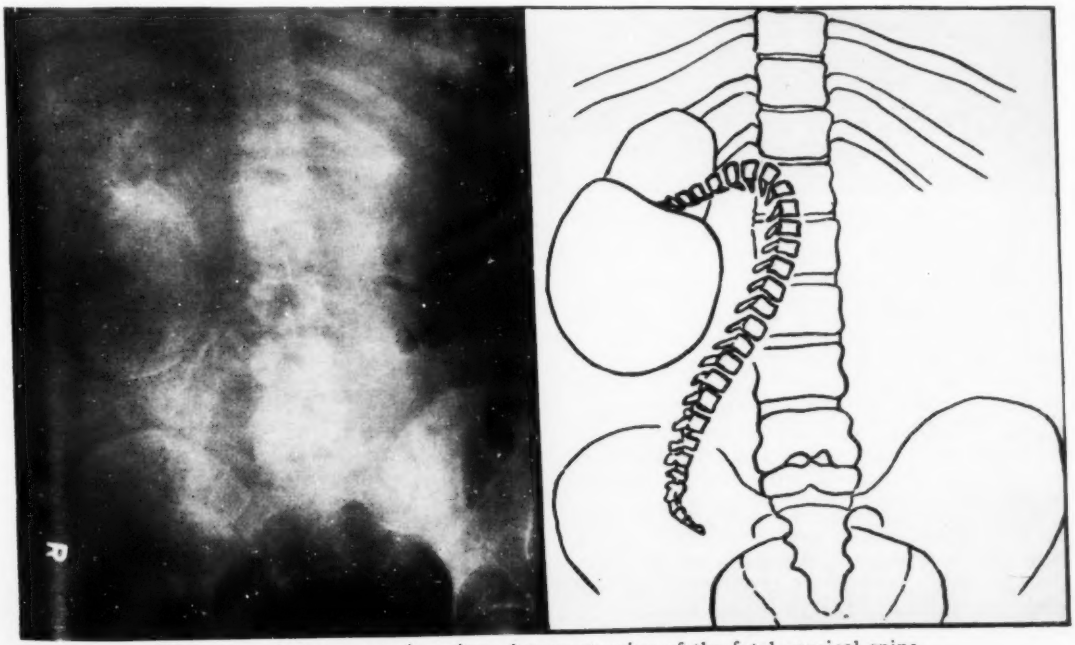

Figure 1 — Antero-posterior view; hyperextension of the fetal cervical spine.

was primary, or whether it was secondary to the abnormal fetal attitude. The normality of the lower portion of the cord suggests that the cervical cord lesion was caused by the fetal cervical hyperextension.

Cases of fetal cervical hyperextension may be divided into two broad groups. There is the group in which the fetus is usually normal and the fault is in the presentation, or lie. These include the face and brow presentation and the "flying fetus". In the second group there is an abnormality of the fetus, such as iniencephaly or a cervical tumour, which results in a fault in the attitude. The case presented does not appear to fit well into either group, being of abnormal attitude, apparently resulting in damage to the cervical spinal cord.

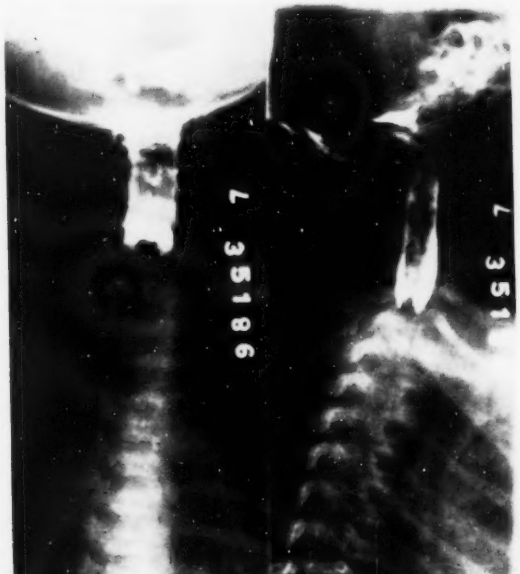

Figure 2 — Antero-posterior and oblique views; obstruction at the level of the seventh cervical vertebra shown by myelography via the cisternal route.

In the first group the most common causes of fetal cervical hyperextension are face and brow presentations (Figure 3). These cases are managed according to the obstetrical principles of the abnormal presentation. Caesarian section is usually necessary with a brow presentation and may be necessary with a face presentation[1].

The "flying fetus" is a form of fetal cervical hyperextension associated with extension of the body and a transverse lie (Figure 4). The head is upright with the face directed laterally, the abdomen directed inferiorly, and the hips extended. The knees are flexed so that the feet approach the lumbar region and the occiput the dorsal aspect of the trunk. This attitude need not be associated with congenital malformation. In any event, delivery can only be accomplished by caesarian section. The hyperextension corrects itself gradually in the post natal period.

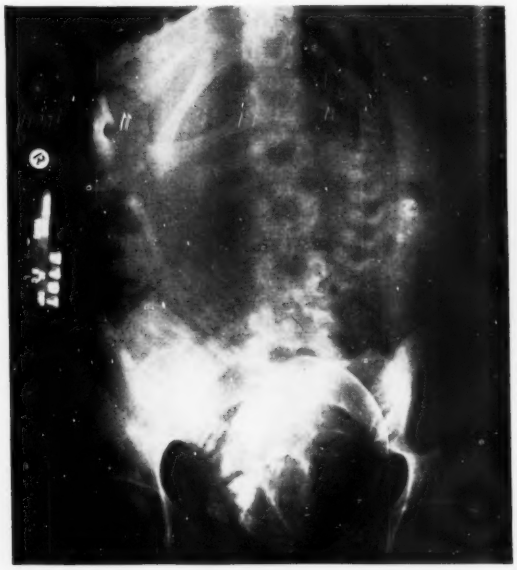

Figure 3 — An antero-posterior view; brow presentation.

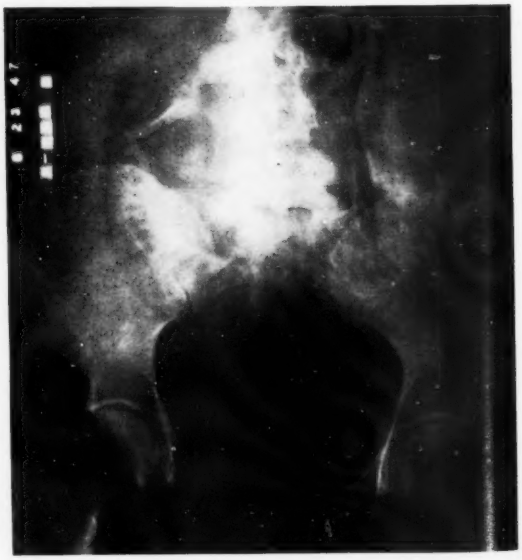

Figure 4 — An antero-posterior view; a "flying fetus", i.e. fetal hyperextension in a transverse lie.

In the second group, an example of maldevelopment associated with fetal cervical hyperextension is iniencephaly (Figure 5). The head, cervical and dorsal spine are extended so that the occiput touches, and to some extent is fused to, the cervical and dorsal spine. The occipital bone is defective and there is some degree of spina bifida in the upper spine. Iniencephaly may be considered a more serious form of the Klippel-Feil syndrome[2]. Delivery can only be accomplished by caesarian section.

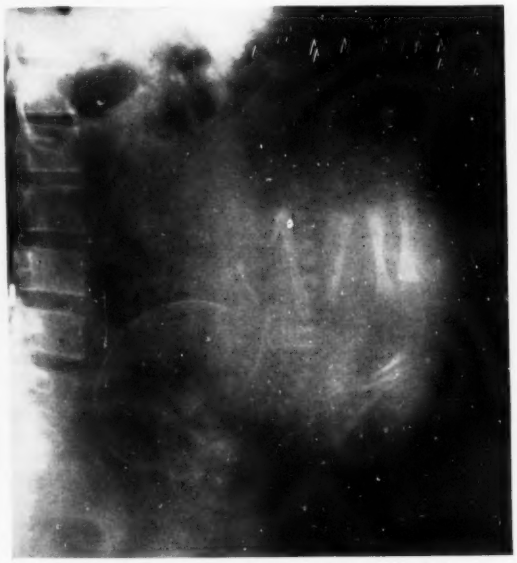

Figure 5 — An oblique view; fetal cervical hyperextension in iniencephaly.

Additional examples of the second group include fetal abnormalities associated with cervical hyperextension due to large fetal tumours of the anterior portion of the neck. These occur in the form of cystic hygroma, branchial cleft cyst, thyroglossal cyst and goitre. Malignant tumours are very rare. It is not likely that these tumours of the neck will be diagnosed or excluded radiologically, but may be suspected. Caesarian section may be necessary.

Although an exact diagnosis cannot be made in most cases, it is important to recognize fetal cervical hyperextension, as it may be associated with one of the serious fetal abnormalities which have been described. With fetal cervical hyperextension one must consider not only fetal abnormality but also caesarian section.

Summary

A case of fetal cervical hyperextension with isolated abnormality of the cervical cord is presented. Fetal cervical hyperextension is discussed as indicative of possible fetal abnormality and as a probable indication for caesarian section.

Résumé

L'auteur présente un cas d'hyperextension cervicale in utero, s'accompagnant d'une anomalie localisée à la moelle cervicale.

Selon lui, l'hyperextension cervicale fétale suggérerait une anomalie possible du fétus et constituerait une indication à une intervention par césarienne.

ACKNOWLEDGEMENTS : The authors wish to thank Dr. J. W. Whitelaw and Dr. P. O. Lehmann for permission to report this case. Thanks is also extended to Dr. R. W. Boyd for help in the preparation of this article.

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BOOK REVIEW

Radiopaque Diagnostic Agents, by Peter K. Knoefel, M.D., Charles C. Thomas, Publisher, Springfield, Illinois, 1961. 140 pages, $6.75.

This is a unique monograph dealing with opacifying substances used in radiodiagnosis. It contains a wealth of information that is only partially obtainable in pharmacologic and radiologic textbooks.

The work is divided into eleven chapters. In the first chapter, the author presents some physical and optical considerations which are fundamental in radiologic work. In the following chapters, the author discusses successively the various radiopaque agents used in the exploration of the alimentary, biliary, and urinary tract, the circulatory system, the respiratory tract, the spinal subarachnoid space, the genital tract, and the reticuloendothelial system. In the final two chapters, the author has grouped miscellaneous procedures and agents, and reviews the problems of toxicity.

In the radiological investigation of each system, the author exposes the inherent problems and discusses the comparative value of the products that are used. This evaluation is based not only on clinical results but also on experimental work, some of which is contributed by the author.

All the compounds used currently and those more recently available in radiodiagnosis, with their official names and their synonyms, are detailed for their chemical characteristics and their clinical behaviour.

At the end of the book, the fatal doses for experimental animals for all these substances are tabulated.

A very extensive bibliography completes this concise monograph. It should be on hand in every radiologic department for consultation by radiologists and other doctors interested in this field.

H.P.L.

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Shaughnessy Hospital, D.V.A.	Vancouver	1152	2	2		Andrew Turnbull
Vancouver General Hospital	Vancouver	1600	8	16		R. W. Boyd
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St. Joseph's Hospital	Victoria	500	6	12		Frank G. Stuart
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Misericordia Hospital	Edmonton	425	4	9		M. Mallett
Royal Alexandra Hospital	Edmonton	884	6	14		C. F. Hyndman
University Hospital	Edmonton	1250	4-5	9		H. E. Duggan
Lethbridge Municipal Hospital	Lethbridge	192		7		M. M. Marshall
St. Michael's Hospital	Lethbridge	183	2	4		Brian O. Black
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Providence Hospital	Moose Jaw	164	1	2		H. O'Rielley
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Holy Family Hospital	Prince Albert	133	2	4		Thomas J. Ho
Medical Arts Clinic	Regina	—	3	6-9		H. P. Kent
Regina General Hospital	Regina	94	4-5	12		A. J. Richards
St. Paul's Hospital	Saskatoon	282	2	4		A. Becker
University Hospital	Saskatoon	525	4	8		E. W. Spencer
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Manitoba Government School	Portage la Prairie	89	11	35		A. W. McCulloch
St. Boniface Hospital	St. Boniface	700	8	16		C. W. Hall
Deer Lodge Hospital, D.V.A.	St. James	715	1-2	3		T. W. Hayter
Grace Hospital	Winnipeg	290	3	6		G. W. Ritchie
Misericordia Hospital	Winnipeg	427	9	9		E. Gedgaudas
Winnipeg Children's Hospital	Winnipeg	232	2	4		A. E. Childe
Winnipeg General Hospital	Winnipeg	862	8	16		R. A. Macpherson
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Brantford General Hospital	Brantford	545	4	6-8		W. E. Crysler
St. Joseph's Hospital	Brantford	168	2	3		J. M. Willinsky
St. Joseph's Hospital	Chatham	211	1	2		J. L. Callaghan
McKellar General Hospital	Fort William	426	4-5	7		A. Molle
South Waterloo Memorial Hospital	Galt	220	3	6		W. R. Bell
Hamilton General Hospital	Hamilton	1100	4	8		T. W. Dean
McGregor Clinic	Hamilton	—	1	3		J. G. Stapleton
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Kingston General Hospital	Kingston	513	8	16		S. L. Fransman
Kirkland and District Hospital	Kirkland Lake	160	1	2		K. C. H. Middlemis
Kitchener-Waterloo Hospital	Kitchener	440	5	8		M. B. George
Ross Memorial Hospital	Lindsay	140	1	2		D. C. Pitt
St. Joseph's Hospital	London	437	7	14		M. B. Hill
Victoria Hospital	London	900	6	12		G. G. Copestake
Greater Niagara General Hospital	Niagara Falls	309	3	6		Margaret E. Bickle
North Bay Civic Hospital	North Bay	100	1	2		T. A. M. Thompson
St. Joseph's General Hospital	North Bay	200	2	5		T. A. M. Thompson
Orillia Soldiers' Memorial	Orillia	120	1	2		T. M. McLennan
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Ottawa General Hospital	Ottawa	622	6	12		Conway Don
St-Louis-Marie de Montfort Hospital	Ottawa	216	2	4		J. A. E. Tessier

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St. Joseph's Hospital	Port Arthur	220	2	4	W. A. Hargan
St. Catharines General Hospital	St. Catharines	400	7	12	G. T. Zumstein
St. Thomas Elgin General Hospital	St. Thomas	378	1	2	W. B. Taylor
St. Joseph's Hospital	Sarnia	345	5	7	
Sarnia General Hospital	Sarnia	247	2	5	G. R. Scarrow
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St. Michael's Hospital	Toronto	950	6	12	E. H. Shannon
Toronto E. Genl. & Orthopedic	Toronto	750	4	8	A. R. McGee
Toronto General Hospital	Toronto	1500	4-8	13	D. E. Sanders
Toronto Western Hospital	Toronto	700	3	7	L. R. Harnick
Welland County General Hospital	Welland	251	1	2	Donald MacNeill
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Hôpital Ste-Croix	Drummondville	180	3	6	Gaston Rodrigue
Hôpital du Sacré Coeur	Hull	320	2	4	Henri Charette
Associated Hospitals of Montreal School of Radiography			7-8	14	Arthur D. French
(Queen Elizabeth Hospital		275			E. M. Crawford
(Jewish General Hospital		384			Isadore Sedlezky
(Reddy Memorial Hospital		139			G. P. Larini
(St. Mary's Hospital		347			J. C. Lanthier
(Montreal Childrens' Hospital		300			J. Scott Dunbar
Hôpital Ste-Justine	Montreal	860	5-7	15	Marc del Vecchio
Hôpital Maisonneuve	Montreal	467	6	10	Jules Laberge
Hôpital St-Luc	Montreal	414	4	8	Ls. Ivan Vallée
Hôtel Dieu	Montreal	750	6-8	15	Albert Jutras
Montreal General Hospital	Montreal	751	8-9	18	D. J. Sieniewicz
Notre Dame Hospital	Montreal	865		20	Yvan Méthot
Royal Victoria Hospital	Montreal	1017	6-8	16	C. B. Peirce
Hôpital du Sacré-Coeur	Montreal	800	4-6	12	O. Raymond
Ste. Jeanne d'Arc	Montreal	479	4-8	12	A. F. Vallée
Hôpital de l'Enfant Jésus	Quebec	607	2	6	Henri Lapointe
Hôpital St-François d'Assise	Quebec	400	2-3	6	J. C. Robitaille
Hopital du St-Sacrement	Quebec	500	2	3	Luc Audet
Hôpital Général St-Vincent de Paul	Sherbrooke	275	5	10	R. L. Duberger
Hôtel Dieu de Sherbrooke	Sherbrooke	400	4	6	André d'Etcheverry
Sherbrooke Hospital	Sherbrooke	150	2	2	John Silny
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Hôtel Dieu de St-Joseph	Campbellton	201	1	1	F. St-Laurent
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Moncton Hospital	Moncton	298	3	6	H. R. Ripley
Saint John General Hospital	Saint John	500	8	16	N. S. Skinner
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Canadian Forces Hospital Affiliated with Canadian Forces Hospitals at Kingston and Rockcliffe, and H.M.C.S. *Naden*, Esquimalt, British Columbia.	Halifax	174		6	Surg. Cmdr. W. M. Little
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St. Rita's Hospital	Sydney	162	3-4	6	H. R. Corbett
Sydney City Hospital	Sydney	233	3	6	H. R. Corbett
PRINCE EDWARD ISLAND					
Charlottetown Hospital	Charlottetown	188	1-2	3	W. L. Macdonald
Prince Edward Island Hospital	Charlottetown	245	1-2	3	W. L. Macdonald
NEWFOUNDLAND					
St. John's General Hospital	St. John's	456	10	20	H. B. Murphy

† Every Director is an M.D. certificated in Radiology or holds equivalent qualifications.

SCHOOLS APPROVED FOR TRAINING RADIOLOGICAL TECHNICIANS IN THERAPEUTIC TECHNIQUE

SCHOOL OR HOSPITAL	CITY	BEDS	STUDENTS TAKEN Yearly	Total	DIRECTOR
British Columbia Cancer Institute	Vancouver	56*	2	3	A. Maxwell Evans
Vancouver General Hospital		20			
Saskatoon Cancer Clinic	Saskatoon		1	4	T. A. Watson
University Hospital		500			
Manitoba Cancer Treatment and Research Foundation	Winnipeg		3	6	R. J. Walton
Winnipeg General Hospital		862			
St. Boniface Hospital		700			
Ontario Cancer Foundation Hamilton Clinic	Hamilton		1	2	L. S. Green
Hamilton General Hospital		25			
Ontario Cancer Foundation, Kingston Clinic	Kingston		1	2	R. C. Burr
Kingston General Hospital		513			
Hotel Dieu Hospital		320			
Ontario Cancer Foundation, Ottawa Clinic	Ottawa		1-2	3	T. G. Stoddart
Ottawa Civic Hospital		36			
Ottawa General Hospital		692			
Ontario Cancer Institute Princess Margaret Hospital	Toronto	137	2-1 yr. course 4-2 yr. course	12	C. L. Ash
Montreal General Hospital	Montreal	751			D. J. Sieniewicz
Notre Dame Hospital	Montreal	865		4	Yvan Méthot
Royal Victoria Hospital	Montreal	1017	2	2	Carleton B. Peirce Jean Bouchard
Saint John General	Saint John	600	1-2	1-2	John A. Caskey
Victoria General Hospital	Halifax	556	2	4	J. E. Stapleton

* In some cases total hospital capacity is given and in others that used for therapy only.

POSITION AVAILABLE

Board Certified or Board Eligible Radiologist required. Salary or partnership basis. Address reply to: Business Manager, The Northwestern Clinic, 220 South Broadway, Crookston, Minnesota.

BOOK REVIEW

Roentgenology of Intracranial Meningiomas, by Sidney P. Traub, M.D., with a foreword by D. L. McRae, M.D., Charles C. Thomas — Publisher, Springfield, Illinois, 1961. $14.00.

Seldom is a book written by a Canadian radiologist. This book therefore should have more than ordinary appeal for the subscribers of this Journal. It has nostalgic appeal for the author of this review since he is pleasantly reminded of many fruitful hours spent with Dr. McRae attempting to master the very subtle details in skull roentgenograms so well demonstrated by Dr. Traub.

The book deals only with the benign meningioma. The clinical records and roentgenograms from The Montreal Neurological Institute of 170 cases of proven benign meningiomas have been reviewed and analysed for this presentation. Some material has been used from The University of Saskatchewan College of Medicine, to supplement the work, especially in the section dealing with cerebral angiography and meningiomas.

The subject matter is presented in logical sequence, commencing with the history, clinical observations, pathologic aspects, change in the skull revealed roentgenologically, pneumoencephalography, cerebral angiography, and finally a summary of the various forms of meningioma, correlated with the anatomic situation of the tumor within the skull.

For the general radiologist, the most valuable part of the book is contained in the first 133 pages. Here are described the changes in skull roentgenograms indicating the presence of an intracranial space-occupying lesion. This is followed by specific evidence indicating the presence of a meningioma.

The roentgenograms are of excellent quality; many reproductions include only that portion of the skull in which the abnormal reaction is situated; this sharpens detail considerably and adds to their value. Appropriate diagrams have been borrowed from other works, mainly that of Cushing and Eisenhardt, to round out the presentation.

One might summarize by saying that a great deal of general neuroradiology has been included in this book as well as specific details related to the meningioma. The bibliography is very complete and arranged alphabetically. 238 pages, 128 illustrations, 135 tables.

D.G.W.